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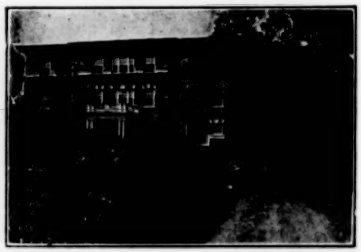

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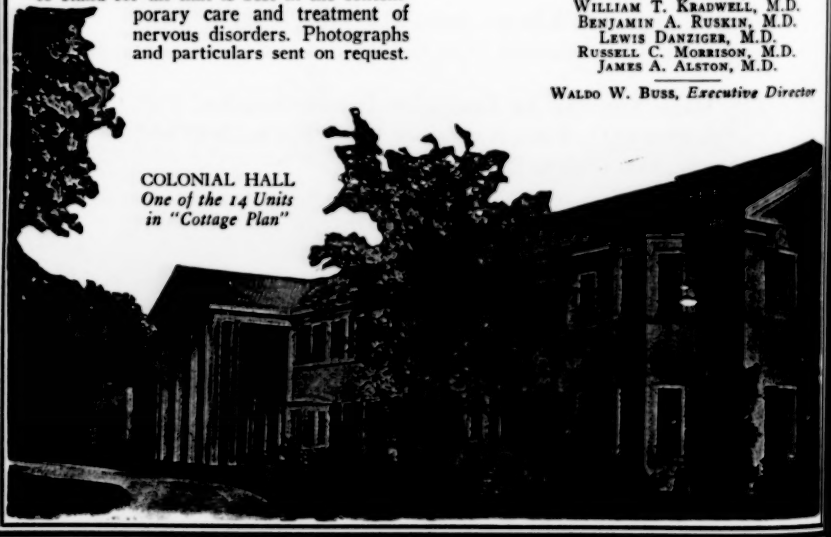

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The Journal
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SOCIOPSYCHOLOGIC FACTORS IN HYSTERICAL PARAPLEGIA*

WILLIAM BROWN, M.D.†
AND
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Eleven patients with hysterical paraplegia were admitted to the Psychiatric Service of the Veterans Administration Hospital, Bronx, New York, between 1946 and 1951. They represent 4.3 per cent of patients with conversion reactions and 0.46 per cent of all patients admitted to the service during this period. The clinical picture of such apparent helplessness and dependency in these young veterans with their "paralyzed" legs, confined to bed or wheelchairs, invites attention to the psychodynamic forces which unite to effect surrender of the ability to walk and the yielding to cravings for aid and support.

Since World War I and especially since World War II a sharp decline has occurred in the incidence of gross musculoskeletal manifestations of hysteria in favor of symptoms related to the vegetative nervous system often manifested by visceral dysfunction. Changes in cultural attitudes, education, group disapproval, and the accusation of "gold bricking" appear to have played significant roles in reducing the number of unmistakable hysterics. In a few cases, however, through identification with true paraplegics, there are apparently florid attempts to act counter to socially approved behavior. A few illustrative cases are cited.

Report of Cases

Case 1.—A 33-year-old white male of Italian extraction, Catholic, single, unemployed, was admitted to the VAH Bronx, New York on Mar.

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5, 1948, complaining of inability to walk and of pain in the back. He bitterly complained that he had injured his back in 1942, after eight months of military service, as a result of lifting a foot locker. Since that time he has had backaches and "heavy legs." He was loud, whining, and truculent in his demand for a pension. In the Army he was hospitalized five times for backache and "paralyzed" lower extremities, and since his discharge in 1945 he was hospitalized in other VA hospitals twice and had had much outpatient department treatment. He had always recovered from his previous attacks of hysterical paraplegia. In the Bronx VAH, after receipt of 100 per cent pension, he was treated for three years to no avail. He received intensive psychotherapy, sodium amytal, hypnosis, and finally a short course of EST and insulin coma. He left his bed and took to a wheel chair and since then has thoroughly identified himself with the paraplegic patients with whom he freely associates. He graduated from public school at 13, worked for two years and suddenly left home, changed his name, and led a nomadic, isolated existence for ten years, traveling about the country, occasionally working and occasionally begging for "handouts." After his alleged back injury in the service in 1942 his buddies "carried" him along, helping him to do his work, massaging his back, and at times even carrying him to the latrine. Since his discharge he has been a burden to his elderly parents. A study of his personality showed repressed hostility, marked passivity, and sexual inhibition. His I.Q. was 117; he was confused as to his sexual role; there was much guilt about masturbation and much fear of expression of hostility. There were evidences of loss of self-esteem with detachment and anxiety. He was discharged from the hospital in a wheel chair, unable to walk, on May 14, 1951, and to date remains unimproved. With government assistance he has had built for himself a home with ramps (as paraplegics do) and he drives a car equipped with manual controls. Apparently he is quite satisfied in his present role. He presents himself to the community as a war hero thereby fulfilling childhood fantasies of success and achievement.

Case 2.—A 22-year-old, single, unemployed, Greek-Egyptian of Greek Orthodox religion. He was in the military service for a period of nine months from September, 1946 until June, 1947. He walked into the Veterans Administration Hospital, Bronx, New York on Feb. 28, 1949, complaining of malaria which he did not have. Two days later he developed hysterical paraplegia. This was his third attack, the first having occurred on Dec. 2, 1948 while attending a television class and the second on Feb. 11, 1949, after slipping on the ice. He recovered from the first attack in six weeks and from the second attack in eight weeks. He told fantastic tales of heroic and daring military exploits, how he was a guerilla in Egypt, a commando in Greece, an officer in the Greek army, a paratrooper in Britain; he said he had won the Victoria Cross and he had made 270 jumps during one of which he had injured his back. None of these tales proved to be true. As far as could be determined from his record he came to this country on Mar. 21, 1946, had served as a private in the Greek army for a few months, and had had five minor courts martial. He was treated with psychotherapy,

sodium amytal, hypnosis with no improvement. He led a wheel chair existence for a few months but his condition remained completely static. He received four EST's with no improvement. On Jan. 2, 1950 he was married in a wheelchair and on Jan. 16, 1950, he left the hospital AWOL. When last seen, April, 1952, he was on television where he participated in a quiz program, "Strike It Rich," winning $500 and claiming that his treatment had failed and that he was in need of money. At that time he was using Canadian crutches. His I.Q. was 89; there were no evidences of psychosis. He was an inadequate individual with strong sex repressions, feelings of inferiority with need for success; he was submissive and conforming and ambitious beyond his ability. He indulged in many fantasies about success but he was fearful of aggression and blamed others for his misfortune. He was constricted, dependent, oversensitive, and craved affection and acceptance. He reacted to emotional stimulation by repression and withdrawal. He was not service-connected.

Case 3.—A 31-year-old white Hebrew male, separated from his wife, was admitted to the VAH Bronx, New York on Nov. 14, 1949, with reactive depression, mutism, and hysterical paraplegia. Prior to admission he had made an attempt at suicide. There were two previous hospital admissions one in 1946 for astasia-abasia and one in 1948 for observation for herniated disc—not found. He was in the military service from January, 1943 until September, 1943. He injured his back on an obstacle course and was discharged C.D.D. There was a long history of psychopathic behavior; as a child he was a behavior problem and was sent to a school for delinquent boys. He served time in the penitentiary for robbery. He had impregnated a 15-year-old girl and was convicted of statutory rape. Later he married this girl but never got along well with her. After a few days in the hospital he regained his power of speech and stated that he had been working in a Navy yard and had struck his head and as a result of this he lost his power of speech and ability to walk. Later he became depressed. He improved slightly as far as his paraplegia was concerned and was signed out of the hospital by his parents in August, 1950. It was discovered later that the patient had charges pending against him. Psychological examination showed an I.Q. of 103, much psychopathy and self-recrimination. There were many guilt feelings as well as strong hedonistic and sensual drives. He indulged in many wish-fulfilling fantasies and showed little constructive capacity. Female figures were rejected. He was subject to rages, was impulsive, and had few assets. He was not service-connected.

Case 4.—A 27-year-old married, white Catholic male, post office worker was admitted to the VAH Bronx, New York on Sept. 13, 1950 with hysterical paraplegia of ten weeks' duration. He had had five and a half years of military service with much combat and many harrowing experiences. On one occasion he was "blown up" by a shell and woke up in the hospital unable to move his legs. He had many guilt feelings about the loss of his comrades. He was sent to Mason General Hospital and treated for a trau-

matic war neurosis with hysterical paraplegia. Since discharge from the military service he has had two or three attacks of hysterical paraplegia every year. Each attack lasted from six to ten weeks. He has been in several VA Hospitals and has had out-patient department treatment. His "paralysis" usually clears up after intravenous barbiturates and hypnosis. Psychiatric examination on this admission showed *belle indifference;* he was co-operative and stated that the attacks had been precipitated by battle dreams. He seemed eager to please and often was ingratiating. During sodium amytal interviews he recounted his battle experiences and was able to move his legs with great effort. With a few further treatments he improved rapidly and was discharged on Nov. 15, 1950.

Discussion

Our study of hysterical paraplegia includes consideration of the life situation of the patients as well as precipitating factors and the clinical course of the illness. This form of paralysis has been observed following trauma, dreams, and after disturbing emotional situations. In his review of the older literature, Binswanger (2) cites a statistical analysis of Briguet who studied 430 hysterics, and found in 120 evidence of complete or partial involvement of the musculature. In 6 cases he reported paralysis affecting the muscles of the trunk and of the four extremities; paralysis of the left upper and lower limbs in 46; the right upper and lower limbs in 14; both upper extremities in 5; the left upper extremity in 7; both lower extremities in 18, and the left lower extremity in 4. Thus, we can see that hysterical paraplegia occurred in 4.4 per cent of these patients. In another series reported by Landouzy there were 370 hysterics of whom 40 showed some type of paralysis, including 14 hemiplegics and 9 cases of paraplegia. In Binswanger's collection of hysterical paralytics one-half showed hemiplegia, one-eighth showed paraplegia, and one-sixth had monoplegia.

The course of this disorder varies with the psychopathology of the patient, his character structure, education, intelligence, inner resources for constructive activity, and the various types of treatment utilized. The symptom generally appears with some suddenness and less often gradually. Di Gaspero (4) inferred that paralysis is frequent after some type of trauma and the onset may supervene hours, days, or longer periods thereafter. In our patients the intervals between trauma and appearance of paraplegia extended from one year to ten years. In 5 cases the interval was six or seven years; the average was six years. Eight of our patients had repeated attacks of paraplegia, each attack appearing suddenly. One attack predisposes to further attacks.

During the "incubation" period, the patient generally mentions subjective vasomotor manifestations, peculiar paresthesias, formication,

numbness of the extremities, falling asleep of the legs, feeling of burning pain in the affected limps, dizziness, headache, flushes, and a sensation of heat. Objectively, there may be such vasomotor alterations as cyanosis, and lowering of the skin temperature. Just before the complete paralysis sets in, the patient may complain of transitory myasthenic sensations, and easy fatiguability. The paralysis which then occurs is always a flaccid one and the sensation in the affected areas is never intact.

Besides motor disturbances, di Gaspero and others have alluded to concurrent implication of psychic processes. There are disturbances in attention, comprehension, and apperception. The memory is labile and somewhat impaired and this feature may be coupled with emotional lability and some tendency to unmotivated explosive, affective outbursts. The sensorium may be clouded and mild degrees of depersonalization may coexist in association with dreamy, sensory distortions. At times the ideation is obsessive and there is a tendency to rapid fatiguability and heightened suggestibility.

The symptom may disappear spontaneously or with a change of situation or environment which precipitated the disorder. Occasionally the disorder may become chronic and at times flexion contractures may set in. This complication occurred with some frequency after World War I. However, a perusal of the "Medical History of World War I" (7) does not specifically make reference to this particular type of hysterical reaction. In the literature one may find case reports describing hysterical paraplegias of many years' duration which yielded to one type of treatment or another. In these cases the psychologic forces had exhausted themselves and the patient, for one reason or another, no longer derived satisfaction from the illness.

In some respects this type of paralysis may be compared with a phobia. In both states there is inhibition of movement: the phobic is progressively restricted in freedom of movement by his fears, each new anxiety-laden situation limiting him more and more until, theoretically at least, he would arrive at a complete psychologic and physical halt. The phobic fears anxiety-producing situations; the hysteric fears these also, but his anxiety is invested in some portion of his body rather than on some external object. The hysterics act as though "my legs refuse to carry me." There is safety in not moving and remaining at a standstill. Abraham (1) mentions that patients with topophobia show a "fear of life" and also a "fear of temptations" which might arise if the person left his protected setting. Ruesch (8) in his discussion of psychologic invalidism mentions that those unable to walk for psychogenic reasons have not been capable of handling their life situations.

Not walking carries the connotation of evading an unbearable dilemma in a face-saving manner. The flight to the dependent position temporarily releases the individual from responsibility, excuses him, superficially at least, from being inferior, and absolves him of the necessity of providing his own support. This is coupled in most cases with a lessening of the need to approach situations in which hostility and aggression might have to be vented.

Human beings react psychologically with certain patterns to feelings of inadequacy, to inability to compete, fear of exposure, fear of failure, inability to form warm interpersonal attachments, and to immature sexual development and inability to be assertive and aggressive. These reaction patterns are determined by cultural and environmental factors and also represent genetic-dynamic influences in the development of the individual. The hysterical reaction which is a frequent consequence of the above-mentioned frustrating situations is generally conceded to have arisen on a psychogenic developmental basis. However, in the older literature, Binswanger mentions that hysterical reactions occur with twelve times greater frequency in individuals of hysterical parents than in those of nonhysterical parents. Apparently the child in his development mimics certain attitudes and imitates the parental behavior which is habitual in a given household.

Certain familial factors, especially those involving separation from the family and the accustomed environment (3), have been thought to play a predisposing role with reference to the origins of the hysterical reaction. The hysteria was found much more frequently in those of rural origin (9), in those of a lower social position, and in those of lower intelligence (6). The older authors found that those who were unusually dependent upon others seemed to present the hysterical reaction with greater frequency.

When decompensation occurs in the adaptive behavior of the individual we observe a return to earlier forms of neuromuscular adaptation. It is not unusual to discover moderate awkwardness and clumsiness in the skillful and purposive movements of neurotics, and with sufficient behavioral disorganization there is further progressive retreat or regression to less highly patterned forms of activity. In enunciating the principle of developmental direction, Gesell (5) has mentioned "the successive head-to-foot sweeps of development." He infers that development occurs in the cephalocaudal direction and twenty-three steps are utilized in the change from prone behavior to standing and walking, a process which takes up to 60 weeks on the average for the infant to complete. This complex integration once attained usually is retained as a lifelong acquisition unless structural damage or neurosis super-

venes. Shaking or wobbly knees, "caving in," various hysterical deformations of the feet and legs, and inability to use both lower extremities are various forms of regression. It is not entirely improbable that the last functions learned, i.e., walking and standing, are the first to respond to the impact of emotional stress. The upright position which is attained with such effort also suffers. People are frequently encountered who are bent by the weight of their problems and conflicts. This may eventuate in the sitting or lying position as the most comfortable adaptation to stress. Cultural determinants with emphasis on the athletic or soldierly stance point to the recognition of erectness in contradistinction to the folded-up posture of the passive-dependent. The utterly helpless individual will resort to some crutch or vehicle for ambulation. Locomotion presupposes goal-directed activity. Intrapsychic helplessness or paralysis arising from the intensity of inner conflict is reflected in the musculoskeletal system as inhibition or loss of locomotion and arrest of goal-directed progress.

Sociologic Factors

Age.—Eleven veterans of World War II were studied. Three were under 25 years of age and 8 were more than 25 years at the time of admission to this hospital. The average age was 26.4 years. The group consisted of 10 men and one girl 27 years of age.

Places of Birth and Residence.—Of the group in question, 5 were born in New York City, and of these, 3 were from Brooklyn. Two patients were born in New Jersey and another in Massachusetts. One was born in Egypt and had resided in Greece before coming to New York and one patient had been born in Russia before being brought to Brooklyn to reside. One was born in Kansas and another in Westchester County, New York. With one exception all had resided in slum or lower middle class areas during the major period of their pre-service life.

Descent.—Four of the veterans were derived from American stock, 2 from Irish parents, 2 from Italian, and 3 from Jewish ancestry.

Marital Status.—Six patients were single and 5 were married; the latter making a relatively poor adjustment to their wives.

Occupations.—These included casual workers, defense worker, petroleum worker, shipyard laborer, lumberman, post office cleaner, vegetable dealer, accountant, and 2 students. The work record could be considered marginal for those in the unskilled group and fairly good in the white collar field.

Schooling.—Four patients attended grade school and two of these graduated; four more attended up to two years' high school and left

for financial reasons; one had attended college for four years and graduated with a B.S. and had been accepted as a medical student. Those with poorer school records were guilty of truancy and showed little interest in their work. Two had made an attempt to obtain some vocational training.

Sports and Hobbies.—Eight showed little or no interest in sports during their school or extramural activities. One was captain of the track squad, and another was proficient in football, swimming, and skiing. Only one patient had a hobby of wood-carving, and was a fairly good artist.

Duration of Employment.—The average duration of any job extended from a few weeks to four years. Most of the patients changed jobs frequently because of their dissatisfaction with employer or type of work.

Military Service.—Of the 11 patients studied, 5 had been overseas and two in combat, and one volunteered for parachute duty. Three cases had less than nine months' service; the one WAC in the series was in the service for 21 months but eight months were spent in hospitals. The remainder of the group had periods of service covering two to five and a half years.

Precipitating Cause.—In the histories of 5 patients, mention was made of injury to the back. One patient strained his back while lifting a foot locker; another had a history of "banging his head and back" with resulting inability to walk or talk; another contused his back falling out of a bunk; still another was hit by a man and fell on his back. Another complained of back pain which was related to an old skiing injury; and, lastly, one suffered back injuries as a result of being "blown up" by a shell. A myelogram and exploratory laminectomy were performed in one case with subsequent onset of symptoms. One patient witnessed his buddy being shot in the back accidentally by strafing Americans with resulting true paraplegia. To escape injury this patient jumped down 15 feet and injured himself. One patient was given a spinal anesthetic in 1942 and in 1946 he had what was diagnosed as encephalomyelitis with moderate quadriparesis. This improved with few residuals but subsequently he became "paraplegic" again. The girl was treated for abdominal pain which was related to subacute gallbladder disease. Walking about the ward she would collapse and fall to the floor, or she would creep along and hold on to the walls. She developed a functional right hemiplegia which improved and this was replaced by the "paraplegia."

It is interesting to observe that with one exception all patients somehow were able to walk to the hospital for admission; their para-

plegias developed after they had been in the hospital for a few days. Apparently there must be some assurance of care and attention before the symptom makes its appearance.

Neuropathic Traits and Habits.—The girl in this series was fat and unattractive; she stuttered and there was considerable sibling rivalry. Among the others may be mentioned such tendencies as nomadism, change of name, delinquency, charges of stealing, and statutory rape, lying and telling of fantastic, absurd tales, truancy, rebelliousness, leaving home at an early age, nail-biting, and enuresis. In one patient there was coprophagia and urinophagia in early childhood. None of the patients partook of alcohol. Only one patient had ever been arrested. This one had been charged with burglary and sent to reform school where he served for 20 months.

Friendship and Sexual Adjustment.—Analysis of the case histories of these patients revealed that most of them had few or no friends and none of them had any really close attachments.

The female patient who was 27, was a virgin and had had few dates with little or no petting. The unmarried male patients had few dates and rare sexual relations with pick-ups. The married men generally made unsatisfactory sexual adjustments. One was impotent and would throw his wife out of bed; another impregnated a girl in an extramarital affair, and only one man made a fair adjustment. There was little evidence of love or appreciation of the partner in any of the patients.

Economic Status of Parental Home.—Seven patients came from marginal middle class homes with considerable financial insecurity and several of the families had been on "relief." In most cases the work records of the fathers were poor. Two patients came from middle class families. Only one patient came from a comparatively well-to-do home, the father in this instance being a cotton broker.

Relations of Parents to One Another.—In only 4 cases were the parents considered to be reasonably suited to one another. In most families there was some evidence of clash and friction; in others there were arguments and recriminations. The father of the patient who served in the reform school was unfaithful and was finally separated from his wife.

Relations of Patients to Parents.—One patient ran away from home at the age of 14 and returned when he was 26 years of age and it is assumed that he adjusted poorly at home. One patient had been neglected by his parents and rebelled with antisocial and aggressive behavior; one patient hated his stepfather; another disliked his father because of the high standards he was expected to reach. Three patients

showed some interest in caring for their parents in some way but the others lived detached from them.

Resort to Wheel Chair and Bed.—All the patients in our series remained in bed for periods lasting from a few weeks to two years. In several the symptoms became stubbornly chronic when they were put to bed and assumed the dependent position. The prognosis was only slightly improved when the patients were transferred to wheel chairs. However, from the point of view of caring for the patient, a wheel chair existence may be considered a vast improvement since the patient could be encouraged to make some efforts toward self-help.

In some ways the wheel chair can be compared to the baby-carriage of the infant—where the caretaker must push the vehicle about. Sadistic and exploitative elements may play a role in the enslaving of a victimized partner or of hospital personnel who must cater to the patient. The wheel chair necessarily evokes expressions of pity and interest toward an individual who might otherwise be neglected and criticized. The patient is also given considerable range of mobility and is no longer required to be helpless and inert in bed. He may travel out of his room and move about the entire hospital with ease and yet at all times will be considered ill without the danger and threat of being regarded as an impostor or malingerer. The picture of the paraplegic is familiar to most lay people who obviously cannot differentiate the vastly more serious organic form from the prognostically more hopeful psychologic form. With improvement and modernization of this vehicle the patient may perambulate readily without help, without inviting censure, and without interfering with the dynamics of his disorder.

Forms of Treatment

All the patients were treated along dynamic or psychoanalytic lines under some form of supervision. Several were treated with sodium amytal or hypnotherapy with some amelioration of the symptoms. One patient, possibly an early schizophrenic, received 60 insulin coma treatments with improvement and 2 patients received short courses of electric shock treatment, because of intercurrent reactive depression. The girl who stuttered received speech therapy and was given beauty care to improve her appearance in addition to the usual psychotherapy.

Diagnosis

Although all the patients exhibited hysterical symptoms, there were other psychopathologic and characterological data to postulate the existence of a character disorder in all but one patient in whom an underlying severely schizoid or possibly schizophrenic process was believed

to exist. Aggressive and antisocial behavior was recorded in the history of several patients. Most of these were detached and seclusive or formed loose attachments to others, with considerable hostility vented whenever there was the slightest frustration.

Psychologic Data.—The I.Q.'s of our 11 patients ranged from 84 to 122; in 5 cases they were below 100. Basically they were all passive, dependent, and submissive individuals. Fundamentally there was fear of hostility, low self-esteem, and confusion with regard to their sexual role. Relations with the maternal figure were often disturbed. Three of the patients showed strong schizoid tendencies manifested by withdrawal, constriction, and narrowing of intellectual and social interests. There was a tendency to overemphasize intellectual capacities at the expense of emotional warmth. All patients showed pronounced immaturity. There were poor interpersonal relationships and in many cases there was little drive to attain reasonable goals. The sexual adjustment was generally poor; often there were strong inhibitions and repressions, feelings of inadequacy, and much guilt over masturbation. One patient had sexual perversions. That the paraplegia served as a defense against hostility and aggression was patently manifest in many of our patients. These ineffectual, immature individuals, seeking vainly for successful accomplishment of long-standing goals, found their efforts toward mature adjustment in sexual, social, and economic spheres obstructed by neurotic conflicts overlying defective character development. Struggles long or short, violent or feeble, to master the difficulties failed and, little by little, a persistent sense of inferiority was developed. Compensatory counter-forces seeking outlets were dampened or completely neutralized by concurrent inhibitory influences. These latter influences developed in early life comprised a pervasive and insistent need for dependency, coupled with basic intolerance or fear of aggression. Numerous symptoms—anxiety, hypochondriacal complaints, emotional outbursts, obsessive phenomena, pseudo-convulsions—seem to be concomitant effects of a one-sided battle with passivity and dependency, finally smothering all opposition and terminating the conflict in theatrical paralytic helplessness. A complete surrender does not always result. The female in our series showed pseudo-convulsive movements and had frequent emotional upheavals. Another patient showed "spasms" of the muscles of the legs; he appeared to be a well of pent-up hostility fearful of its release. With his generalized spasticity and immobility and his grim facies, he resembled a picture of suppressed or "frozen" rage. Not infrequently it seemed as if the patients have a kind of poorly defined realization of their incapabilities and resort to childlike grandiose fantasies. Many of the patients showed passive sexuality

with fear or avoidance of women. In one case there were powerful repressions against latent homosexuality. There was much evidence of impulsive, explosive, attention-seeking behavior. Fear of punishment and guilt, especially in relation to autoerotic practices were common findings.

Treatment and Prognosis

Eight of our patients recovered from their paraplegias or were sufficiently improved to walk out of the hospital. Three patients (Cases 1, 2, and 5) were discharged in wheel chairs, unimproved; these individuals all had had extensive periods of hospitalization (in Case 1 for 38 months), but intensive psychotherapy, physiotherapy, and all varieties of ancillary treatments were of no lasting benefit. Seriously defective character development in these cases was an outstanding feature largely responsible for the refractoriness to therapeutic attempts. Follow-up studies (as of June, 1952), with the exception of Case 6, whom we cannot trace, disclose that no recurrences have supervened in the improved or recovered patients. Case 6 was a nomadic individual who had had 16 admissions to various V.A. hospitals throughout the country. His attack of paraplegia always seems to be precipitated by oncoming winter and there was always rapid improvement in the spring. He had many schizoid features (*see* Table).

It must be borne in mind that all except one of the patients developed the paraplegia in the hospital. Early diagnosis and prompt treatment may forestall a chronic outcome. The longer the paraplegia persists, the poorer is the prognosis. Secondary gain derived from the illness becomes an important factor. Those who are service-connected for a psychiatric disorder may be awarded additional compensation plus extras for their families. This extra income continues only as long as they remain in the hospital and thus a security is obtained which has not been granted them in private industry. While in the hospital, in addition to psychotherapy, these patients are literally smothered with gifts, outings, parties, shows, and special attention from various service organizations, attention which they would never obtain away from the hospital. All this generally reinforces the desire to remain in the hospital. Paradoxical as it may appear, solicitous treatment in comfortable surroundings encourages in these patients a greater dependency. The incentive to re-establish themselves economically outside the hospital therefore may never become very strong. Early discharge and vigorous out-patient treatment in terms of rehabilitation may improve the prognosis, even if they leave the hospital in wheel chairs.

In individuals whose hysterical paraplegia is superimposed on seri-

TABLE I.—Results of Treatment of Hysterical Paraplegia in Relation to Military Service, Duration of Hospitalization, Service-Connection, and I. Q.

Case	Military Service	Duration of Hospitalization (Mo.)	Service Connection	Type of Paraplegia	I. Q.	Condition on Discharge	Follow-up
1.	Oct/41–May/45	38	S. C.	Recurrent	117	Unimproved	Wheelchair existence at home
2.	Sept/46–June/47	11	N. S. C.	Recurrent	89	Unimproved	Uses crutches at home
3.	Jan/43–Sept/43	9	N. S. C.	Recurrent	103	Moderately improved	Uses crutches or cane
4.	Aug/40–Mar/46	2	S. C.	Recurrent	115	Recovered	Working; no recurrence
5.	Jan/45–Apr/47	29	S. C.	Recurrent	110	Unimproved	Hospitalized elsewhere
6.	Oct/42–May/43	5	S. C.	Recurrent	115	Recovered	Whereabouts unknown
7.	Sept/42–May/45	1½	N. S. C.	First attack	99	Recovered	Asymptomatic; no recurrence
8.	Sept/43–June/45	5	N. S. C.	Recurrent	98	Recovered	Working; no recurrence
9.	Mar/41–Aug/45	2	S. C.	Recurrent	92	Recovered	No recurrence
10.	Sept/42–May/46	5	S. C.	First attack	122	Recovered	Working; no recurrence
11.	Aug/43–June/45	8	S. C.	First attack	84	Much improved	Uses cane; working; O. P. D. treatment

ous character disorder we must acknowledge the probability of a poor prognosis. These patients do not readily acquire insight and their response to the usual forms of therapy is not likely to be favorable. Hysterical symptoms are notoriously apt to recur under conditions of stress unless the basic character structure is changed. It is not easy for these individuals to master their problem of dependency and concomitant hostility, and effect more wholesome interpersonal attachments. Lifelong, deeply patterned rebellious behavior and attitudes, whether expressed or suppressed, would in most cases render difficult the acceptance of therapeutic suggestions in the direction of conformance and compliance, that is developing and seeking rational economic goals, more fruitful social intercourse, and a healthy sexual attitude. Ruesch in his study of delayed recovery from various illnesses finds that associated emotional problems exert a retarding effect on the total rehabilitation. The inability of the patients to accept or tolerate interpretations with regard to these emotional factors is an essential reason for the failure to improve.

The association and fraternization of hysterical paraplegics with true organic paraplegics (as may occur in this hospital) may thwart therapeutic efforts in the case of the former. The permanence of symptoms, sympathetic pity, and liberal financial benefits in the case of true paraplegics appear quite attractive to the hysteric whose mechanism of identification is easily aroused. Therefore it is clear that contact between the two types of patients should be discouraged and if possible avoided.

As a long-term project the hysterical paraplegics must receive some type of vocational or occupational rehabilitation. If they can be taught some occupation in which they may take pride and obtain a fair financial return, there will be some incentive for constructive employment.

A genuine acceptance of a dynamic interpretation of the illness leading to basic insight and consequent improvement is uncommon in these cases. They are often unable to grasp the significance of their disturbed interpersonal relations. In order to achieve a continued state of satisfying adjustment in all areas these individuals must be made aware of their failures in social, sexual, and economic spheres and they must be impressed with a sense of responsibility to participate wholeheartedly in therapeutic programs. As long as they remain emotionally blunted to the feelings of others and to their unresolved conflicts, the prognosis remains in doubt. These patients do not readily think dynamically and they often are relatively impervious to the true meaning of psychodynamic explanations. There appears to be a selective blindness to their problems and frequently it is only with prolonged analytic treatment that a therapeutic impress is made on their capacity to deal with

problems requiring conscious participation and mature judgment.

How to motivate these patients to get out of bed, or to forsake their wheel chairs before their identification with paraplegics becomes too fixed, remains a therapeutic question. The tendency to be permissive to these patients has resulted in chronocity. Firmness tempered by understanding would seem to be more applicable in these cases. In a general way a therapeutic program should be directed toward attempts to re-educate the patient to activity through the aid of physical rehabilitation. In every possible manner efforts must be made to increase the patient's self-esteem. Time-honored methods of heat, massage, passive and active exercises, use of the swimming pool should all be utilized to induce the patient to exert spontaneous efforts in his own behalf. Supervision should be kindly but vigilant, and there should not be the slightest intimation that the patients are pretending or "putting on" or "gold bricking." Since the patients may reject a psychologic formulation of their illness, they might be more receptive to an explanation of their illness as an expression of some type of organic malady which will respond to physical therapy. This relieves them of any guilt, stigma, and responsibility in relation to their participation in the genesis of their disorder. The patient, especially if he has a low I.Q., will often vehemently deny that his disability is a consequence of his life attitudes and will seek to maintain his self-esteem through the mechanism of attributing his symptoms to spontaneous structural changes over which he has no control.

Summary

Eleven cases of hysterical paraplegia were studied from the sociologic and psychologic viewpoints. The dynamics of these cases were elucidated and factors responsible for chronicity were outlined. The frequent coexistence of severe character disorder rendered the prognosis doubtful in many cases. Particularly significant was the refractoriness to psychotherapy and the inability to accept dynamic interpretation. In those cases which did not respond to psychotherapeutic efforts, physical rehabilitation seemed the more effective form of therapy.

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NOSOLOGIC POSITION OF NEURASTHENIA IN PSYCHIATRY

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The term neurasthenia was first introduced by George M. Beard in 1868 before the New York Medical Journal Association. His first paper dealing with the subject was published in the Boston Medical and Surgical Journal, April 29, 1869, and a second paper in June 1878, in the Virginia Medical Monthly.

In his treatise on nervous exhaustion (neurasthenia), published by E. B. Treat, 1888, Beard (p. 157) summarized his views on neurasthenia by stating that it is "a chronic, functional disease of the nervous system, the basis of which is impoverishment of nervous force, waste of nerve-tissue in excess of repair, hence the lack of inhibitory or controlling power physical and mental, the feebleness and instability of nerve action and the excessive sensitiveness and irritability, local and general, direct and reflex. The fatigue and pain that temporarily follow excessive toil or worry, or deprivation of food or rest, are symptoms of acute neurasthenia from which the chronic form differs only in permanence and degree. Nervousness is really nervelessness."

The major symptoms described by Beard as characteristic of neurasthenia are approximately 53.

To clarify the nosologic status of neurasthenia in the light of present psychiatric knowledge it is necessary to list these symptoms:

Symptoms of Neurasthenia, According to Beard

1. Tenderness of scalp
2. Dilated pupils and sudden and frequent alterations between contraction and dilatation
3. Sick headache and various forms of head pains
4. Pain pressure and heaviness in the back of the head and over the vertex
5. Changes in the expression of the eye, hard to describe
6. Congestion of the conjunctiva
7. Disturbances of the nerves of special sense (neurasthenic asthenopia or irritable eye)
8. Muscle volitantes or floating specks

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9. Noises in the ear (tinnitus aurium, sudden explosions or pulsations)
10. Atonic voice
11. Flushing and fidgetiness
12. Insomnia
13. Drowsiness
14. Tenderness of the teeth and gums
15. Nervous dyspepsia (*dyspepsie asthénique*)
16. Deficient thirst and capacity for assimilating fluids
17. Desire for stimulants and narcotics
18. Abnormalities of secretions
19. Abnormal dryness of the skin, joints, and mucous membranes
20. Sweating of hands and feet, with redness (palmar hyperidrosis)
21. Salivation (not so common)
22. Tenderness of the spine (spinal irritation)
23. Coccygodynia
24. Heaviness of the loins and limbs (of the myelasthenic variety)
25. Shooting pains simulating those of ataxia
26. Podalgia (pains in the foot)
27. Tremulous and variable pulse and palpitation of the heart (irritable heart)
28. Local spasms of muscles (tremors)
29. Convulsive movements (spasmodic movements) especially in going to sleep
30. Dysphasia
31. Cramps
32. Special idiosyncrasies in regard to food, medicine, and external irritants
33. Sensitiveness to changes in the weather
34. Localized peripheral numbness and hyperesthesias
35. A feeling of profound exhaustion
36. Ticklishness
37. Vague pains and flying neuralgias
38. General and local itching (pruritus)
39. General and local chills and flashes of heat
40. Cold feet and hands
41. Nervous chills
42. Sudden giving way of general or special functions
43. Temporary paralysis
44. Diseases of men (involuntary emission, partial or complete impotence, irritability of the prostatic urethra)
45. Diseases of women (uterine and ovarian irritations)

46. Oxalates, urates, phosphates, and spermatozoa in the urine
47. Gaping and yawning
48. Rapid decay and irregularities of teeth
49. Deficient mental control
50. Mental irritability
51. Frequent blushing
52. Hopelessness
53. Morbid fears

The last five symptoms were purposely catalogued by me at the end of the tally because they represent in my estimation the mental component of the syndrome, as described by Beard.

In Beard's own words deficient mental control (49) represents "the inability to concentrate the intellect on any task, as in writing or thinking. The mind wanders away in every direction and when, brought back by an effort of will, is liable to be soon lost again in reverie. In some cases the exercise of concentration or even slight attention is exceedingly irksome and painful, causing distress."

Mental irritability (50), according to Beard, consists in the fact that "the patient finds himself in a condition to fret and worry, becoming irritable over trifles. The flurries of domestic life, the cares of the house, disappointments and vexations, become a source of great distress."

Under the heading of frequent blushing (51) Beard stresses the point that "some young men are so harassed by this symptom that they cannot meet young ladies in the street or in company of their own set without blushing excessively; and on this account they frequently keep away from society altogether."

Concerning hopelessness (52) the author states that the patient "even in the earlier and milder stages is without hope, while the friends laugh at his fears. The philosophy of this symptom of hopelessness appears to be similar to that of morbid fear, an instinctive consciousness of inadequacy for the task before us."

Morbid fears (53) are considered by Beard as "psychical functional paralysis." The patient knows that there is no just objective ground for his fear, but his emotional nature, under the influence of his exhausted nervous condition, overcomes his reason and will. Among the morbid fears Beard mentions the following: astraphobia (fear of lightning), agoraphobia (fear of open square, public places), topophobia (general fear of places), claustrophobia (fear of narrow, closed spaces), antropophobia (fear of mingling with a multitude or of meeting anyone), monophobia (fear of being alone), pathophobia (fear of diseases), pantophobia (fear of everything), phobophobia (fear of fears), mysophobia (fear of contamination), etc.

One readily realizes what an intricate clinical picture neurasthenia was, in Beard's original conception and formulation; a mixture evidently of somatic and mental manifestations with no attempts at separating or integrating mental and somatic elements. Least of all was there any attempt made at establishing the primacy of the psychic or somatic manifestations. The pathogenesis was to be found in the "impoverishment of nervous force and waste of nerve-tissue in excess of repair."

Even after the concept of neurasthenia was accepted, particularly under the sponsorship of such authorities as Moebius and Erb in Germany, of Hutchinson, Flint and Campbell in England, of Grasset, in France, and of Rosenthal in Austria, confusion existed as to the nosologic value of such a pleomorphic syndrome.

Arndt, in his textbook on neurasthenia (1885), maintained that neurasthenia is not a clinical entity but the result of various etiologic factors, somatic and psychic, "acting upon a nervous system which first tires easily and later on exhausts itself. The nervous system, hindered in its normal development, maintains embryonic and foetal properties, so that it reacts to stimuli with all the characteristics of the immature tissue."

Among the clinical manifestations of neurasthenia, Arndt described mental symptoms under the form of what he calls "psychic hyperesthesia" (melancholy) and "psychic hypoesthesia" (mania). Anxiety and phobias were part and parcel of neurasthenia.

In 1893, Von Hösslin, in Müller's handbook on neurasthenia, emphasized the psychic components of neurasthenia especially the variations of mood, depression, and agitation, to which he added compulsive ideation. Von Hösslin discussed in some detail the symptom "anxiety" which in neurasthenia may appear as anxiety-like reactions (scrupulosity, timidity), free-floating anxiety, and the anxiety of the phobias (agoraphobia).

The emphasis on anxiety in neurasthenia is found also in Kaan's paper on "Der Neurasthenische Angstaffeckt bei Zwangsvorstellungen" (1893), and elaborated on by Hecker in his contribution "Ueber larvierte und abortive Angszüstande bei Neurasthenie," dating from the same year.

It was in 1894 that Freud wrote his paper on "The Justification for Detaching from Neurasthenia a Particular Syndrome: The Anxiety Neurosis." The name was proposed because all of its component elements could be grouped, according to Freud, "round the central symptom of morbid anxiety and because individually they each have a definite connection with this." He included both anxiety neurosis and what was left of neurasthenia under the heading of true neurosis attributing to masturbation the main etiologic factor for neurasthenia,

and to "the withholding of the somatic sexual tension from the psychical and its interference with its elaboration in the psychical field, for anxiety neurosis." Both neurasthenia and anxiety neurosis were considered dependent on a somatic factor; in neurasthenia its was impoverishment of excitation and in anxiety neurosis an accumulation of excitation. Both were considered not amenable to psychotherapy. Neurasthenia and anxiety neurosis constituted the group of the true or actual neuroses, whereas hysteria and obsessional states constituted the group of the psychoneuroses.

I will not enter here into a discussion of the etiology of neurasthenia but will proceed with the historical review of the concept of neurasthenia as it developed chronologically in various countries after Freud's contribution.

In Krafft-Ebing's textbook of psychiatry (1904 translation) we find that for this author neurasthenia or nervous weakness covered a state of the general nervous system characterized by the two fundamental symptoms of abnormal impressionability and extreme exhaustibility, the expression probably of disturbed nutrition of the central nervous system. He added: "neurasthenia is a general neurosis, i.e., one affecting the whole nervous system, and since mental disturbances play an important role, it may be called a 'neuropsychosis' occurring in a benign form in persons without predisposition and due to a temporary and remediable cause." Among the mental symptoms Krafft-Ebing spoke of "painful feelings of mental inhibition, deficiency in mental ability, anxious feeling of serious diseases, emotional irritability, erethism of the cortex, as a result of which the imagination becomes intense and certain thoughts especially those of painful character are constantly presenting themselves."

Krafft-Ebing made no distinction between neurasthenia and anxiety neurosis. Anxiety determined by outstanding symptoms such as cardiac irregularities was considered part and parcel of neurasthenia.

In 1915, Kraepelin, having mentioned the possibility that the basis of neurasthenia could be found in a nervous exhaustion, some sort of chronic or metabolic poisoning, ends by stating that "everything was guess work" and that he was not able at that time to prove anything exactly.

Hertz, in Bumke's Handbook of Psychiatry (1928), described neurasthenia as a basic "irritable weakness of all nervous functions." He enumerated a series of mental and somatic symptoms with no particular mention of anxiety or attempt at separating the anxiety neurosis from the neurasthenic syndrome. Phobias were not mentioned in his description.

In Switzerland, Dubois (1910) strongly emphasized the psychogenic aspects of neurasthenia. He expressed himself as follows: "I must point out two drawbacks to the word neurasthenia. It takes the supposition that it is the nerves that are not equal to their task, as if it were a matter simply of a physico-chemical alteration as yet unknown, of the nervous elements. This conception has taken possession of the minds of medical men; it is current among the public and it has caused the psychological influences to be forgotten, the considerable part which the mind plays in the genesis of these states."

For Dubois the physical asthenia is only one facet of neurasthenia. "Whether one is dealing with the incurable invalids, who remain all their lives in this state of true neurasthenia, with the less rebellious neurasthenias, which last for some years, or with the frequent cases which yield rapidly to a rational psychotherapy, it is necessary always to put in the foreground the psychic symptoms: the pusillanimity, the tendency to discouragement and sadness, and the absence of self-mastery.

"The neurasthenics bring into their activity indecision and scruples derived always from fear. It is this insufficiency of the intelligence particularly in the ethical domain that one has to recognize as the primary trouble . . . There one sees the fundamental psychopathy where neurasthenic crisis is only an episodic manifestation provoked by debilitating influences. There is already some psychasthenia in these patients, there is already some degeneration; they are not of the strong."

The appellation psychasthenia, according to Dubois, should be applied to the patients in whom the phobic obsession, the tics, the manias, etc., predominate. Here the psychasthenic state is evident, the mental synthesis is particularly defective.

Contrary to Freud, Dubois felt that psychotherapy is the therapy of choice for neurasthenia. Psychotherapy should follow the lines of educating the patient along moral ethical lines to fight his pusillanimity and want of self-mastery.

In Bleuler's textbook of psychiatry (1924), neurasthenia is included under the heading of "neurotic syndromes" (hysteria, neurasthenic syndrome, expectation neurosis, compulsive neurosis, and accident neurosis).

Bleuler divides neurasthenia into neurasthenia proper and pseudo-neurasthenia. Proper, or actual neurasthenia, or chronic nervous exhaustion is of physical origin and has preponderant physical manifestations. In neurasthenia there is an exhaustion of the nervous system as a result of a long continued exaggerated activity or as a result of normal activity in the frame of congenital morbid exhaustibility.

Pseudo-neurasthenia is of psychic origin, and, because of all its symptoms and its purely psychic suggestibility, is as much of a mental disease as hysteria. It is comparatively more frequent than the nervous exhaustion and represents a definite form of flight into disease, which for itself and others, shams (unconsciously) the nervous breakdown and for this reason so closely resembles the nervous exhaustion. Bleuler did not differentiate the group of the anxiety neurosis with its characteristic phobias as originally described by Freud. He discusses the phobias under the heading of compulsive neurosis.

R. Brun (1950) has advanced a conception of the neuroses as "hormopathies," after the Greek word "horme," instinct, impulse. He divides the hormopathies into organic hormopathies and functional hormopathies.

The organic hormopathies include:

I. Dysglandular (endocrine) hormopathies
II. Constitutional endogenous hormopathies
    (a) endogenous psychoses
    (b) congenital psychopathies
III. Cerebral hormopathies

The functional hormopathies include:

I. Functional organic (toxic) hormopathies
   Actual (vegetative) neuroses
    (a) neurasthenia
    (b) anxiety neurosis
    (c) fright neurosis
II. Psychoneurosis in the strict sense of the term (conversion neuroses)
    (a) hysteria or hysterical reactions
    (b) phobias (anxiety hysteria)
    (c) obsessional neuroses

Brun considers neurasthenia an "irritable weakness that is, on the one hand, an intensified susceptibility to almost all external and internal stimuli, and, on the other hand, an abnormally rapid incidence of fatigue of the nervous system, as the result of any kind of effort, whether physical or psychical; briefly, an abnormally ready response (lowering of the threshold of excitation) to all cerebrospinal and vegetative excitations, combined with abnormally rapid fatigue in respect of motor output."

In neurasthenia "psychical primary symptoms" are never entirely

absent and they too obey the law of irritable weakness. They consist of increased affective lability, enhanced responsiveness to affects and lability of affects; in the early appearance of mental fatigue; in a certain inability to concentrate, and finally, in a tendency to depression. These psychical primary symptoms, according to Brun, should not be confused with the "complex conditioning" psychic processes of an unconscious character operative in the psychoneurosis.

Apart from the difficulty of differentiating between primary psychic symptoms and conditioned psychic symptoms, Brun seems to increase the difficulty of separating neurasthenia from the psychoneuroses when he accepts masturbation as one of the etiologic factors in neurasthenia.

The difficulty stems from the fact that Brun does not agree with the concept of somatic dangers in masturbation. He feels that the psychic concomitant circumstances are of much greater importance. In his views, the injurious effect on the vegetative nervous system occurs in connection with the painful feeling of guilt (whose real content is always ultimately unconscious) and the chronic anxiety awakened thereby causing the most intensive excitations of the sympathico-adrenal system. Where does he draw the line between somatic factors, psychical primary symptoms and complex conditioning psychic processes?

In France, Janet considered neurasthenia as the starting point for the development of psychasthenia or "maladie des obsessions." Neurasthenia was, for Janet, "a vague state of weakening or lowering of vital functions and in particular of the central nervous functions corresponding to a physiological and anatomic substratum as yet unknown in its essence."

The hereditary predisposition constitutes the background for the development of neurasthenia; secondary factors, especially intoxication and autointoxication, the latter particularly pronounced at puberty, play an important part in determining neurasthenia. However, neurasthenia is, for Janet, a condition which manifests itself through both physiologic and psychologic symptoms. The physiologic symptoms are related to the dysfunction of the cardiovascular, gastrointestinal, and pulmonary systems. The psychologic symptoms include abulia, aprosexia, indifference and apathy, all manifestations of what Janet terms diminution or lowering of the function of reality. This diminution of the function of reality which is the expression of a lowering of psychologic tension, can be observed in various degrees. In neurasthenia, the lowering of both nervous tension (somatic) and psychologic tension (psychic) has no particular characteristics. It is some sort of undifferentiated lowering of tension. Gradually, this lowering of nervous and psychologic tension

develops into a more definite and characteristic type of mental disorder —the obsessive state of psychasthenia.

This development occurs by the addition to the neurasthenic state (lowered nervous and psychic tension, and its resulting lowering of the sense of reality) of other symptoms which Janet calls "feelings of incompetence" (feelings of inadequacy). From then on, with the complications resulting from somatic symptoms and development of anxieties and phobias, the full picture of psychasthenia, as conceived by Janet, is established. Neurasthenia is therefore not an autonomic disease but a mild form of psychasthenia.

For Dejerine and Gauckler neurasthenia is a psychoneurosis. By their definition "it is constituted by a general ensemble of phenomena, which result in the nonadaptation of an individual to any continued emotional cause, and the struggle of the individual to bring about such an adaptation."

"We lay it down," they add, "as a general rule, that there is always an emotional cause in the genesis of neurasthenic states. If you cannot find such a cause, it is because either your diagnosis is at fault, and that your patient is neither an hysteric or a neurasthenic, or else your patient is deceiving you."

Among the somatic manifestations of neurasthenia, Dejerine and Gauckler include manifestations involving the cardiovascular, respiratory, digestive, motor, and sensory systems. To these they add the symptoms of anguish. Anguish is for them a physical feeling which "corresponds to the psychic feeling of anxiety." It sometimes consists of bodily sensations of thoracic constriction, with the sensation of smothering, sometimes of feelings of dull, deep-seated, boring or stabbing pain, which is frequently localized at the pit of the stomach and which may become crystallized in the form of nervous pain.

In the United States, W. White (1935), classified neurasthenia under the heading of "psychoneuroses."

The fundamental symptom of neurasthenia is fatigability, both mental and physical. With the fatigability there goes a condition of irritability, irritable weakness, and inability to concentrate the attention for any length of time. "The mood is pessimistic, often hypochondriacal. The simple depression that goes with this state is often of the nature of a symptomatic depression. The hypochondriacal ideas referable to the particular viscera are to no small extent fostered by the treatment. To the condition of these patients' continuous attention to the functions of their body organs, the name hypochondria is given."

For White, idleness, especially in women, is at the base of the fact that the "thoughts naturally turn to self and in this factor is laid the

foundation of the hypochondria that these patients suffer from, the external complaining of little nothings. Having lost their touch with reality, and become self-centered, their ego occupies a too prominent place in their perspective."

In connection with therapy, White felt that "this group of cases are especially favorable for a rational psychotherapy. . . . The rational approach to these problems (awakening new interests and training them gradually to healthy viewpoints) is via the psychoanalytic method."

Yet when White discusses the problem of anxiety neuroses, he states that, unlike hysteria, anxiety hysteria, and the compulsion neuroses, both anxiety neurosis and neurasthenia hypochondria, do not have their origin in the psychic sphere but in the lack of harmonious adjustment of the physiologic functions of the sexual organs.

Schilder (1930) stated that neurasthenic and hypochondriac symptoms are projections from the psychic sphere of central interest to another sphere which is at least felt as somatic and does not belong to the nucleus of the personality. It is felt on the periphery of the self.

Conversion, for Schilder, not only takes place in hysteria but also in neurasthenia and hypochondria. Neurasthenia and hypochondria, like other neuroses, are ways of getting rid of tormenting conflicts. Their mechanism is one of conversion which tries to keep closer touch with reality than the hysteric mechanism. Hypochondriac and neurasthenic symptoms must therefore have an unconscious meaning. "By constitutional or acquired weakness in hypochondria and neurasthenia, single parts of the postural model of the body are chosen out of the whole body picture and attract more libido than they should. Neurasthenia and hypochondria are diseases where the fixation of the libido to the different parts of the postural model has been directed to particular spots (fixation at the narcissistic level)."

In contrast to the definitely psychogenic origin of neurasthenia expressed by Schilder, true neurasthenia for Wechsler is an organic disease in the sense that as yet undemonstrable changes are the cause of the symptoms and not the result of psychogenic processes.

A. Brill, discussing the topic of neurasthenia at the same symposium with Schilder and Wechsler (1930) concluded that after looking through his records carefully, he could not find a single case that he could diagnose as a typical neurasthenia. "Neurasthenia as it is understood by the average physician hardly exists . . . even if we restrict the term to the symptoms left in Freud's classification of the neuroses." Brill for himself was never able to diagnose neurasthenia.

Strecker and Ebaugh consider neurasthenia a psychoneurosis and

describe as its symptoms most of the original somatic ones described by Beard, in addition to feelings of inferiority, irritability, depression, phobias, and anxieties.

Muncie (1939) classifies neurasthenias in the group of the psychoneuroses, Meyer's "merergasias" (from *meros,* part) or "kakergasias" (from *kakos,* bad).

Noyes (1948) also includes neurasthenia under the heading of psychoneuroses. He states that in neurasthenia the concern that really belongs to psychic problems, frustration, and conflict is transferred or displaced to an uneasiness of mind concerning physical organs and processes.

Weiss and English (1949) classify neurasthenia under the heading of neurosis which includes also conversion hysteria, anxiety hysteria, and compulsion neurosis.

In England, Henderson and Gillespie (1950) describe neurasthenia under the heading of psychoneurosis, though for "accuracy its use should be confined to those cases in which fatigue, mental or physical, is the prominent symptom." Therapeutically, by implication, neurasthenia is amenable to psychotherapy, inasmuch as "it represents with the psychoneuroses, a faulty response to the stresses of life and especially to those inner tensions that come about from confused and unsatisfactory relationships to other people."

In Italy, Tanzi and Lugaro considered neurasthenia a component of what they termed "obsessive psychosis." In neurasthenia all symptoms are of psychic origin and they represent a variety of obsession—"obsession of exhaustion." Gozzano considers neurasthenia a psychoneurosis and among its symptoms he includes the various phobias. Ottonello emphasizes in neurasthenia such psychic factors as variation of mood, emotional hypersensitivity, underestimation of one's own potentialities, doubts, fears, and worries.

We thus see how confused is the concept of neurasthenia, how uncertain are its boundaries, how neurasthenia is considered by some as an organic disease or an actual neurosis not amenable to psychotherapy, and how it is considered by others as a psychogenic disorder with characteristics of transposition, displacement, and symbolism.

Clarification is needed. I feel that we must discuss at this point the arguments in favor and against the concept of neurasthenia as a psychiatric clinical entity still deserving its individuality after the clinical syndrome of anxiety neurosis has been detached from it.

In order to evaluate the problem better, let us consider what Freud described under the heading of anxiety neurosis, a syndrome which he

originally detached from neurasthenia. According to his original paper (1894) here are the symptoms of what he termed anxiety neurosis.

(1) General irritability.

(2) Anxious expectation; for one form of anxious expectation, that relating to one's own health, Freud reserved the old term of hypochondria.

(3) Anxiety (not usually present in consciousness) can erupt suddenly into consciousness and thus bring about an anxiety attack. An anxiety attack of this kind consists of a feeling of anxiety alone without an associated idea, or associated with the nearest interpretation, such as sudden death, a stroke, or approaching insanity. Or else the feeling of anxiety is combined with paresthesias (similar to the hysterical aura); or, finally, together with the feeling of anxiety there is an accompanying disturbance of any one or more of the bodily functions such as respiration, heart action, vasomotor innervation, or glandular activity.

(4) The degree to which these elements are combined in anxiety attacks varies extraordinarily and almost every accompanying symptom can *alone* constitute the attack just as well as the anxiety itself can. There are consequently rudimentary anxiety attacks and equivalents of an anxiety attack. These are as follows:

(a) Attacks accompanied by disturbances of the heart action such as palpitation, with arrhythmia or tachycardia
(b) Attacks accompanied by disturbances of respiration (nervous dyspnea, attacks similar to asthma and the like, not always accompanied by recognizable anxiety)
(c) Attacks of sweating, often nocturnal
(d) Attacks of tremor and shuddering
(e) Attacks of ravenous hunger
(f) Attacks of sudden diarrhea
(g) Attacks of locomotor vertigo
(h) Attacks of so-called congestion, practically embracing all that has been called vasomotor neurasthenia
(i) Attacks of paresthesias

(5) Awakening in fright (pavor nocturnus).

(6) Vertigo. Its mildest form is better described as giddiness and its graver manifestations attacks of vertigo (with or without anxiety) constitute one of the most momentous symptoms of this disease.

(7) On the basis of chronic apprehensiveness (anxious expectation), on the one hand, and a tendency to attacks of vertigo with anxiety, on the other, two groups of typical phobias develop, the first relating to common physiologic dangers (fear of snakes, thunderstorms, darkness,

vermin, and so on), the second related to locomotion—agoraphobia, with all its accessory forms collectively characterized by their relation to movement.

(8) Nausea and biliousness, tendency to diarrhea.

(9) Paresthesias—atypical and changing and even a tendency to hallucinations.

(10) Just as an attack of vertigo may be replaced by a fainting fit, so may chronic giddiness be replaced by a constant feeling of sinking to the ground, exhaustion, etc.

If now we were to remove from the original description of Beard's neurasthenia all the symptoms described by Freud under the heading of "anxiety neurosis," we would clearly appreciate the fact that actually nothing is left to characterize the clinical picture of neurasthenia. Freud himself contributed to this feeling when he confined the clinical designation of neurasthenia to the few remaining symptoms of "headaches, spinal irritation, dyspepsia with flatulence, and constipation."

It is, in my estimation, the combination of psychogenic and somatogenic symptoms that characterized the original concept of Beard's neurasthenia. To deprive a syndrome of one of its main components (the mental one), to reduce it to the simple expression of a few somatic symptoms, and yet force its inclusion in the field of psychiatry as a clinical entity or even syndrome is an untenable position.

Freud's distinction between neurasthenia and anxiety neurosis has been kept alive in most textbooks of psychiatry, especially in the United States and England, as a result of which the description of neurasthenia is made under a separate heading immediately preceding the description of anxiety neurosis or anxiety hysteria. However, while most authors still cling to Freud's concept of neurasthenia as a separate entity, when it comes to the description of clinical examples one cannot help thinking that what is described under the heading of neurasthenia is really nothing but a case of anxiety neurosis as originally conceived by Freud.

We must therefore question once and for all the contradiction of differentiating from neurasthenia the clinical picture of anxiety neurosis, while still keeping alive a skeleton structure of a few somatic symptoms under the heading of neurasthenia.

It is my belief that there are no fundamental or substantial differences between the clinical picture first described by Beard under the heading of neurasthenia, and that described by Freud under the heading of anxiety neurosis.

Freud's concept of separating from neurasthenia the clinical entity of anxiety neurosis, leaving to neurasthenia the few somatic symptoms

often mentioned previously, can hardly be reconciled with his statement that "from the anamnesis of patients we frequently find that the symptoms of anxiety neurosis have at some definite time supplanted the symptoms of some other neurosis, for instance neurasthenia, and have taken their place."

At what stage of neurasthenia does the change occur, and what are the symptoms of neurasthenia which are supplanted by those of anxiety neurosis? Asthenia? flatulence? dyspepsia? constipation?

On the other hand, how are we to visualize the coexistence of the two syndromes or entities in the same patient, as Freud maintains when he states that there are cases of mixed neuroses in which neurasthenia and anxiety neurosis are both present: "A man who had previously been in the habit of masturbating and had become neurasthenic becomes engaged to be married and is sexually roused during the intimacy with his fiancée, acquires anxiety neurosis in addition to his neurasthenia."

Anxiety neurosis being a clinical syndrome characterized by outstanding somatic and mental manifestations must, in my estimation, survive. Its etiology and psychodynamics justify it.

In another paper dealing with the nosologic position of anxiety neurosis in psychiatry, I have advocated the maintenance of the phobias as part and parcel of the symptoms of anxiety neurosis proper and the discontinuance of the term anxiety hysteria created by Freud to define specific phobias.

Neurasthenia stripped of any substantial mental component, even of the elementary affect of anxiety—the absence of which was the justification for Freud's creation of anxiety neurosis—should be denied a nosologic position in psychiatry.

This does not mean that the somatic symptoms of neurasthenia will not be encountered in practice. Fatigue, tendency to exhaustion, somatic symptoms referable to dysfunction of some vegetative function may still be found as the major complaints of individual patients.

In the presence of such a condition one must think in terms of a dysfunction which belongs to the field of internal medicine, neurology, or endocrinology.

We are here in the presence of a somatic condition presumably the expression of a vegetative disturbance or imbalance which could deserve the designation of "vegetative neurosis."

Such a vegetative neurosis need not belong at all to the field of psychiatry unless anxiety sets in. If this association occurs, the vegetative neurosis should be qualified as "associated with anxiety," and only under these circumstances could the syndrome be assigned to psychiatry.

In the pure form of somatic or vegetative neurosis the syndrome could be compared to the "cenesthopathies" described by Dupré in 1907. Under this name Dupré described a dysfunction of the "kenesthesis," i.e., of the common general sensibilities (from *koinos,* common, general, and *aisthesis,* sensation). The cenesthopath, according to Dupré, complains of disturbances of the internal perceptions of the general tonus and of the synthesis of general sensibilities especially the vegetative ones. Dupré does not extend the meaning of the word cenesthopathy to changes in the mood and affective or emotional tonus.

The cenesthopath complains of vague, ill-defined tension, heaviness, swelling, distention, constriction, discomfort, etc., in various areas of his body. These annoying, persistent sensations, almost mysterious as Dupré puts it, are even difficult to express verbally. They do not correspond to a definite pathologic entity.

The cenesthopaths do not consult the psychiatrist. They visit the family physicians who, according to the predominance of the symptoms, refer them to various specialists. These in turn, being unable to find any objective justification for the complaints, refer the patients to the psychiatrists who end by making a diagnosis of neurasthenia, hypochondria, or hysteria.

Dupré objects to these various diagnoses and emphasizes his concept that cenesthopathy is a primary disturbance of the general common sensibilities related to the vitality and functioning of our internal organs. For Dupré, cenesthopathy is a primary condition related presumably to constitutional abnormalities of somatic sensibilities.

Cenesthopathy may be present in a pure form independently from any other psychiatric manifestation. However, and this is of interest to us, Dupré states that hypochondriacal ideas, anxiety states, and depressions may develop as secondary complications.

Without any further delving into the clinical picture of cenesthopathy as conceived originally by Dupré, I should like, in the light of our modern knowledge of anatomy and physiology of the autonomic nervous system, to qualify the cenesthopathies as "vegetative neurosis."

A vegetative neurosis, i.e., a dysfunction or imbalance of one or more vegetative functions, may delevop on a constitutional basis of inadequacy, structural or functional, of any portion of the intricate mechanism of endocrine-vegetative integration, and may be viewed as a constitutional autonomic inadequacy. In such cases, the prognosis would be poor.

On the other hand, inadequacy, dysfunction, or imbalance of the endocrine-vegetative system may be of an acute and more or less transitory nature, in which case appropriate therapy may prove beneficial, and the prognosis favorable.

Reference is made to the cenesthopathies in support of my impression that neurasthenia, deprived of all the symptoms of anxiety neurosis, does not differ from the concept of cenesthopathy as conceived by Dupré, a condition which I propose to label as a vegetative neurosis.

Clinically, neurasthenia would therefore represent a somatic situation which, in the light of more recent knowledge, could be considered a somato-neurosis, a vegetative neurosis, constitutional or acquired, not belonging to clinical psychiatry but to the field of internal medicine, neurology, or endocrinology.

ELECTROENCEPHALOGRAPHIC STUDIES DURING THE COURSE OF INSULIN COMA TREATMENT OF SCHIZOPHRENIA*

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Introduction

Davis and Davis (4) in 1939, studied the records of 232 patients including 93 schizophrenics, 64 manic depressives, and 9 psychotics with cerebro-arteriosclerosis, 7 with epilepsy, and 41 "miscellaneous." They concluded that psychotics, as a group, had a significantly larger percentage of abnormal electroencephalograms than did normals. P. Davis in 1940 (5), classified electroencephalograms of schizophrenic patients into four groups. Group I was those who were mostly paranoids, and had normal records. Group II showed dysrhythmic patterns like those seen in epilepsy, its variants or equivalents. Group III had "choppy" patterns on the electroencephalograms which suggested the possibility of localized or generalized irritative lesions, cysts, atrophy, etc. Group IV records showed features of both Groups II and III. P. Davis (7) later, in a comparative study of schizophrenics and manic depressive patients, stated that 50 per cent of the records of 126 schizophrenic patients evaluated showed fast frequencies. Low voltage records were said to be more frequent among psychotic patients. "Choppy" records were seen in patients with schizophrenic deterioration. It was pointed out that mescaline, which also produces a choppy record in electroencephalograms, produces symptoms not unlike those of schizophrenia. McNeel et al. (21) said that the "quality" of electroencephalograms of schizophrenics is consistently lower than that found in the normal population.

Jasper and his group (18) in 1939 pointed out that many schizophrenics showed electroencephalograms similar to epileptic patterns. Lemere (20) in the same year, stated that schizophrenic records show a relative deficit in alpha activity and there is a greater tendency for delta activity especially in malignant and deteriorated cases. Finley and Campbell (8) found the greatest number of abnormal records in the catatonic and hebephrenic group, with the paranoid cases showing more

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nearly normal records. They felt that there was nothing consistent or diagnostic in the electroencephalograms of schizophrenic patients.

A large series of 1,593 cases was studied by Greenblatt (11) and 23 per cent of the records of schizophrenic patients in this group were called abnormal as against 10 per cent abnormal records in a control group of normals. However, Greenblatt pointed out the greatest number of abnormal records occurred in the youth and old age, with the lowest number in the age group of 25 to 45 years. Electroencephalographic changes then, in psychotic patients as in normals, were considered largely the function of the age of the patients.

Cohn (2) stated that the electroencephalographic abnormalities are infrequently seen in institutionalized cases of dementia praecox.

There are few electroencephalographic studies in the American literature on insulin coma treatment. In 1937, Hoagland et al. (17) did electroencephalographic studies on 6 schizophrenic patients during 35 separate insulin comas. The emphasis of this study was on the individual treatment period and it was shown that there was a progressive decline in alpha frequencies from 60 to 70 per cent of pre-treatment values during production of the coma. No conclusions were drawn in this study as to the effect of the electroencephalogram on the aggregate of insulin coma treatments. The decline in alpha frequencies during each insulin coma was considered to be directly proportional to the rate of carbohydrate metabolism of cells producing the rhythmic cortical pattern. (The influence of carbohydrate level on brain activity was shown also to be a measure of oxygen supply (9). The reciprocal relationship of carbohydrates and oxygen supplies and their reflection in alpha rhythm of the electroencephalogram has been confirmed (14).)

Davis (5) in 1940, reported a study of 16 patients who received "pharmacological shock" (insulin or metrazol), and stated, "The electroencephalograms of these patients, if normal before treatment, become abnormal during and following the termination of 'pharmacological shock.' Patterns abnormal before treatment remained abnormal and abnormalities increased during and following the termination of treatments." This was felt to be an indication that insulin coma treatment exerted a harmful effect on the brain. However, no distinction was made between this type of treatment and Metrazol in evaluating these findings.

McNeel (21), et al. did electroencephalograms before, during, and following insulin coma treatment on 33 patients and found that it was not possible to observe "deteriorative changes in the cortex" as determined by the electroencephalogram.

Hoagland, Cameron, and Rubin (16) suggest that the electroencephalogram may be used to prognosticate patient's course during insulin coma treatment since they say that electroencephalographic changes occur prior to any other objective symptomatic changes.

Knott and Gottlieb (19) studied 10 patients before a series of "coma-producing" insulin treatments and repeated electroencephalograms between three and 12 days after the last coma period. The records in all but 2 patients became more "normal" in that the alpha index increased following treatment.

Materials and Method:

A continuous series of 50 patients treated with insulin coma treatment was studied by means of serial electroencephalograms. In this series, there were 41 patients with diagnosis of paranoid schizophrenia, 2 with catatonic schizophrenia, 3 with hebephrenic schizophrenia, 2 schizophrenia unclassified, one schizophrenia mixed type, and one emotional instability reaction with marked schizoid features. These were all healthy young adult males; the age range was 20 to 38 years with an average of 27.8 years. In this group of patients, there was no history of head trauma, cerebrovascular disease, encephalitis, or of convulsive disorders except in one case. This patient gave a history of encephalitis presumably due to influenza and occurring at an early age. The patients in this study had had symptoms of their illness for one to three years before hospitalization with the onset of the acute break with reality occurring within a few weeks of hospitalization.

A six-channel Grass electroencephalograph, using conventional leads, was employed. Electroencephalograms were performed before the institution of a course of insulin coma treatment, and repeated regularly at intervals of three to four weeks, following recovery from a regular insulin coma period. The records were made following either breakfast or lunch and blood sugars were drawn on all patients who showed abnormal recordings. All such blood sugar determinations were in normal range, 80 to 120 milligrams per cent. At least five tracings were taken on each patient during the course of treatment. A follow-up electroencephalogram was performed within one month following the last coma period with a longer post-treatment observation period when indicated. All records were first studied individually and then comparisons made with others taken serially during the study.

The alpha index (6) was determined on the pre-treatment and post-treatment records of 26 patients who showed sufficient improvement to be discharged within 90 days following insulin coma treatment. The index is defined as the amount of elapsed time during which countable alpha waves are present in a meter sample of record. In this study, the

alpha index was independently determined by three examiners on two separate meter length samples. From this, an average mean was determined and then a statistical analysis was performed to determine whether the individual scorers could reliably compute the alpha index.†

The records were analyzed by means of the Chi Square Test of Significance, the hypothesis being that the differences occurring between the scorers would be no greater than those which would happen by chance alone. It was found that there were real discrepancies between the three scorers, with the over-all Chi Square for the 26 records being significant at beyond the .001 level of significance. In only five records could it be reasonably assumed that the differences between the scorers were not significant.

Because of the relatively high dispersion in the distribution of the alpha index determinations, it was felt that a more valuable method for establishing reliability would be to rank the patients in a one-two-three order according to alpha index values, with the highest value receiving the number one position. The differences between these rankings were calculated and the correlations between the three scorers were found to be .89, .90, and .90, indicating moderately high reliability in determining the alpha index.

These reliability tests indicate that a patient can be reliably ranked in a group for the relative alpha index values and it would seem advisable that interpretations be made on the basis of the rank order rather than the actual percentage of alpha waves. In other words, all patients showing a high percentage of alpha waves were consistently placed in a high position by the three scorers and those receiving a low percentage were consistently placed in a low position. Apparently, the qualitative aspects affecting the determining of percentages do not affect the relative position which the patient holds in the group.

Insulin coma treatment was carried out according to the technique described by Bond and Shurley (22). The smallest maintenance dosage required to produce hypoglycemic coma level to Stages III and IV (15), was 70 units. The highest maintenance dosage was 350 units. The course of treatment constituted a minimum of 40 hours of deep coma (Stages III and IV), up to a maximum of 60 hours of deep coma depending on the clinical response of the patient.

Findings

In the present series of 50 patients, careful inspection of the pre-treatment records showed 47 tracings were normal, i.e., they had a

† The statistical studies were carried out by members of the Psychological Department under the direction of Leah M. Loehrke, Ph.D., Chief Clinical Psychologist, Veterans Administration Hospital, Cleveland, Ohio.

frequency of 8½ to 12 waves per second and an amplitude range of 10 to 50 microvolts. Seven of these normal records were of the low-voltage, fast type, showing a frequency between 16 to 24 per second and a voltage ranging between 8 to 16 microvolts persistently in all leads. The fast frequency records were not similar to the "choppy" records described by P. Davis. According to the Gibbses (10), 11.6 per cent of the normal population show this type of record. Hyperventilation performed during the taking of each of these recordings for a period of two minutes produced no significant changes in the tracing. One record which was considered abnormal showed a basic frequency of 24 to 28 per second with a few runs of five to seven per second and this occurred in a patient with a past history of encephalitis. The other two abnormal records each showed runs of slow activity of five to seven per second frequency in both frontal and parietal leads. In these 2 cases, there was no previous history of a disease process which would account for the findings. At least 10 per cent of the normal population show such a record.

The general survey of the electroencephalograms in this series shows then an over-all "normality." This may be related to the type of patient studied since most of the group fell into the classification of "paranoid" schizophrenia and patients with "deteriorative" changes were not selected for insulin coma treatment. This group then could be considered to correspond most closely with P. Davis' (5) Group I mentioned above.

In addition, a detailed study of 26 of these records was made by means of the alpha index. The system of classification of alpha frequencies of Davis and Davis (3) was employed. In this study, the distribution range of alpha index in normal adult individuals was found to be as follows:

25 per cent Rare (16-24 per cent alpha)
20 per cent Mixed (25-49 per cent alpha)
35 per cent Subdominant (50-74 per cent alpha)
20 per cent Dominant (75-100 per cent alpha)

In this series, the distribution of alpha index determined on 26 patients was:

16 (61.5 per cent) Rare
5 (19.2 per cent) Mixed
4 (15.4 per cent) Subdominant
1 (3.9 per cent) Dominant

It is seen then, that a disproportionately large number of the records (61.5 per cent) fell into the "Rare" group when compared with the

Davises' normal distribution curve above. It must be remembered that the present series is relatively small, however, and this factor may influence the fiindings.

In 17 of the 26 cases in which alpha index was determined, there was no significant change in the index before, during, or after insulin treatment. Of the remaining 9 cases, 5 showed a decrease in alpha index by more than 10 per cent. In 3 of those cases which showed a decrease, a change from Mixed to Rare group was noted. In one instance, there was a change from Dominant to Subdominant and in another from Subdominant to Rare. In those cases showing an increase in alpha index during insulin coma treatment, 2 changed from Rare to Mixed group and the other 2 from Rare to Subdominant group.

In the over-all study of over 250 electroencephalographic records taken on these 50 patients, no persistent abnormalities appeared as a result of insulin coma treatment. During the treatment period of these 50 patients, the following descriptive detailed changes in frequency, amplitude, and the effect of hyperventilation were noted:

(A) Changes in Frequency: In 14 cases, no alterations in frequency or amplitude occurred in the electroencephalogram as a result of the insulin coma treatment. In addition, there were 6 cases of low voltage fast activity, likewise considered normal, which persisted throughout and following the treatment.

In 2 cases, a slight increase in frequency from an average of 9 per second to 12 per second and with an average amplitude of 70 millivolts, appeared. This change persisted in only one case in the post-treatment record. In one case, the low voltage fast record appeared during the treatment period and persisted in the follow-up study. In 3 other cases, a low voltage fast record appeared only in the recordings made after treatment had been completed.

In 6 patients with a pre-treatment normal record, each showed a record with prominent runs of abnormal slow waves averaging 4 to 6 per second on at least one routine examination during the treatment period. In one patient, the slow wave activity was present in a recording taken the following week. These changes were always bilateral and most prominent in fronto-parietal areas. All of these cases, however, at the next regular interval of testing, showed no further evidence of abnormality. One of these abnormal records appeared on the day in which a convulsive seizure occurred during insulin coma treatment. Records kept of all hypoglycemic convulsive seizures indicated that there was no correlation between the number of seizures and abnormality of the electroencephalogram. One patient had 20 hypoglycemic convulsive seizures during his course of treatment without developing

any abnormality in any of the records obtained. Unpublished data (13) shows that the electroencephalogram is of no value in forecasting the occurrence or frequency of convulsive seizures which occur during the hypoglycemic period of insulin coma treatment. There were no abnormalities to be noted following hyperventilation in this group of cases.

(B) Changes in Amplitude: In five records which were normal before treatment, there was an increase in amplitude during or following treatment though not to a degree which could be considered abnormal. In three of these, an increase of 20-30 microvolts was noted during treatment. In two of these three, the amplitude continued to rise 20-30 microvolts more in the post-treatment record. In the two other serial records of these five, there was a gain in amplitude of 20-40 microvolts in the post-treatment record, although this change was not seen in any of the previous records. In these five records, the most marked gain in amplitude occurred in the occipital areas.

Three normal pre-treatment records showed a decrease in amplitude of 40-50 microvolts during treatment. This decrease persisted in the post-treatment record in one instance and in the other two, the records showed a low voltage fast pattern.

(C) Changes in Hyperventilation: Five normal pre-treatment cases showed changes in hyperventilation during the course of insulin treatment. However, the routine runs revealed no specific alteration which could be considered significant. In each of these cases, re-examination at the usual interval did not again reveal this finding. These hyperventilation changes, without the accompanying changes in the routine run, from clinical experience, carry limited significance. One case, during the second month of treatment and when repeated the following week, showed runs of 4-6 per second frequency, and on hyperventilation revealed a large build-up which persisted for more than a minute after termination of hyperventilation. An electroencephalogram repeated at the usual interval did not reveal any abnormality in the spontaneous run or on hyperventilation. Another case on one occasion, in the eleventh week of insulin treatment, showed a rare run of 4-6 per second activity, and on hyperventilation showed a moderate build-up of significant nature which, when repeated three weeks later, was not present.

Abnormal Records. One abnormal pre-treatment record which contained prominent runs of 24-28 per second frequency and also rare runs of six per second frequency, changed during the treatment period to show a slow record with six per second waves predominating, occurring mainly in the frontal and parietal areas. This slow wave activity appeared in every routine tracing and persisted for three months following insulin coma treatment when the last record was obtained. This case

also showed a large build-up on hyperventilation in each recording obtained. There were two other patients who showed abnormal slow wave activity obtained prior to treatment which did not again appear during or following treatment.

Prognostic Value of the Electroencephalogram. This serial study of all electroencephalograms taken during the course of insulin coma treatment revealed that the electroencephalogram has little value in prognosticating the therapeutic effects of insulin. A total of 26 out of 50 patients were discharged from the hospital within 90 days following completion of treatment having showed sufficient social adaptability to make an adjustment in the community. In this group of 26 patients, the serial electroencephalograms of 17 maintained a normal appearance throughout the period of observation. There were 3 cases which appeared normal in the pre-treatment records which, during the treatment period, showed variations in frequency and amplitude which were within normal limits. The post-treatment records in these cases likewise were within normal limits. The variations in these cases were recognized only on serial comparison of the records.

Of the remaining cases in the "Normal" group, the following observations were made: There were 4 cases which showed runs of abnormally slow activity on one occasion during all of the serial studies, but which did not appear again in subsequent recordings. (These records will be discussed at another point.) One patient's record in this group appeared abnormal prior to treatment and continued abnormal throughout the period of observation. Another patient's record showed abnormal slow activity prior to treatment but on subsequent examination during and following treatment, the record appeared normal in all respects.

Discussion

The pre-treatment electroencephalograms of these 50 patients showed no characteristic electrical pattern. The tracings appeared, on gross inspection, to be within the normal range of frequency and voltage and without the usual signs indicative of abnormality. A study of the alpha index in 26 of these pre-treatment records showed a variation from that found in the normal population. This finding is in agreement with Lemere.

A study of the electroencephalograms of these 50 patients revealed that the accumulative effect of at least 40 hours of deep insulin coma produces no progressive or persistent abnormalities in the electroencephalographic tracings. This work is in keeping, therefore, with the finding of McNeel (21) who found no "deteriorative changes" produced by insulin coma treatment as determined by electroencephalograms. On

the other hand, the serial study of these cases did not corroborate the findings of Hoagland et al. (16) that the electroencephalogram may be used as a tool to prognosticate the clinical results of insulin come treatment. In the 26 patients whose improvement could be ascribed to insulin treatment, there were 23 normal pre-treatment records; the variations which did appear gave no evidence that the changes were related to the improving clinical status of the patient.

A study of the alpha index of this group of 26 patients indicated that about 65 per cent (17 cases) showed no change in the alpha activity. In only 19 per cent (5 cases) was there a decrease and in 15 per cent (4 cases) was the increase of significant degree. It would seem that the alpha index did not reflect clinical improvement as noted by Knott and Gottlieb (19).

Those cases which showed abnormal slow tracings on one or more occasions (in one instance) during treatment were of a temporary nature not unlike those found in a previous study (12) dealing with transient neurologic complications during insulin coma therapy. The changes in frequency which appeared in individual records no doubt resulted from altered but reversible cerebral metabolic function. The changes within a normal range of frequency which occurred in two instances cannot be considered significant and came to light only on reviewing that particular series of tracings. In those 2 cases where a decreasing trend in the voltage was noted, the post-treatment record showed a low voltage fast record. Whether this indicates a disturbance in the cortical pattern is open to conjecture. In none of the records did the amplitude reach a degree which can be considered abnormal. Here again, only on serial comparison, did the increase or decrease in amplitude become recognized and its significance appeared negligible. In the one record which appeared grossly abnormal prior to, during and after treatment, it appeared that the insulin coma treatment at least accentuated the abnormality originally seen.

While a build-up following hyperventilation itself is not pathognomonic of abnormality, it may gain added significance if other signs of electrical disturbance are present in the electroencephalographic tracing. In five instances, where a build-up did develop during treatment, it was not considered significant since the routine electroencephalogram was normal in all respects. In 2 of the cases which showed some slow runs which alone were not sufficient to be considered abnormal, there were significant changes on hyperventilation to conclude that the record was at least beyond the borderline of normal. The build-up in these 2 cases was more prominent in those areas of the brain in which the slow runs appeared. This corresponds with the findings of Cohn (1)

who noted that during electric shock treatment, likewise, hyperventilation increases the prominence of a slow wave output. In our cases, a return to the normal electrical patterns was noted at the next regular recording. These changes apparently are reversible and appear as the result of temporary individual metabolic stress for similar reasons as ascribed above for the slow-wave records obtained.

It is apparent that insulin coma treatment influences the electroencephalogram quite differently from electric shock treatment. The findings would indicate that the majority of records continue within a normal pattern and are of no help in making an evaluation of the clinical improvement of the patient. The electroencephalogram, it appears, has its most pertinent value in corroborating the neurologic status resulting from a temporary disturbance of cerebral chemistry from insulin coma treatment.

Conclusions

1. A group made up of predominantly paranoid schizophrenic patients showed no significant or characteristic electroencephalographic pattern with the use of conventional leads.

2. A course of insulin coma treatment produces no cumulative disturbance in the electrical pattern of the electroencephalogram.

3. Determination of alpha index showed that these records had a relatively low amount of alpha activity.

4. The alpha index proved to be of little value as a prognostic tool in predicting or evaluating clinical improvement of the patient.

5. The electroencephalographic abnormalities seen during and following an individual coma treatment were transitory and reversible.

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THE HYPOPHYSIS CEREBRI IN PSYCHOSIS*

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AND
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We are presenting our findings of living organisms, as seen under the phase microscope in the hypophysis cerebri from 100 patients who were afflicted with mental illness. We have found that the hypophysis was subject to an infestation with same kind of zoospore organisms as were previously found in the brain. A special technique was devised for making suspension preparations of the gland.

Material and Technique

Equipment used in preparing saline suspensions for examination with the phase microscope was sterilized. It was put into 5 x 9 inch instrument trays with covers. Each kit contained 3 knives, 3 forceps, 2 small Stender dishes for specimens, 1 dropper, 1 sheet of aluminum 3 x 8 inches on which to place instruments when in use, 1 glass 5 cc. syringe with a 20 gauge hypodermic needle for Vaseline. The trays were sterilized in an autoclave. Sterile normal saline in a dropper bottle was on hand. Wide microslides, 78 mm. x 38 mm. and cover glasses, 24 mm. square, were sterilized separately, as were the Vaseline and rubber gloves. Ultraviolet radiation was used from time to time on the microscopic benches.

The hypophysial suspensions were prepared in the following way. The gland was removed with sterile instruments, placed in a stender dish, and brought to the laboratory. A small drop of normal saline was placed on several slides. The neural part was incised and some of its contents were placed in the drops of saline. They were gently pressed to form a turbid suspension. This was covered with a coverglass sealed to the microslide with Vaseline. Then the distal part of the gland was cut into halves. The cut surface of the gland was dipped two or three times in the drop of saline on the microslide to form a turbid suspension. The secretory cells and living organisms dropped out readily. The coverglass was pressed down, and sealed to the microslides with warmed sterile Vaseline. Preparations of pars intermedia were made by using a

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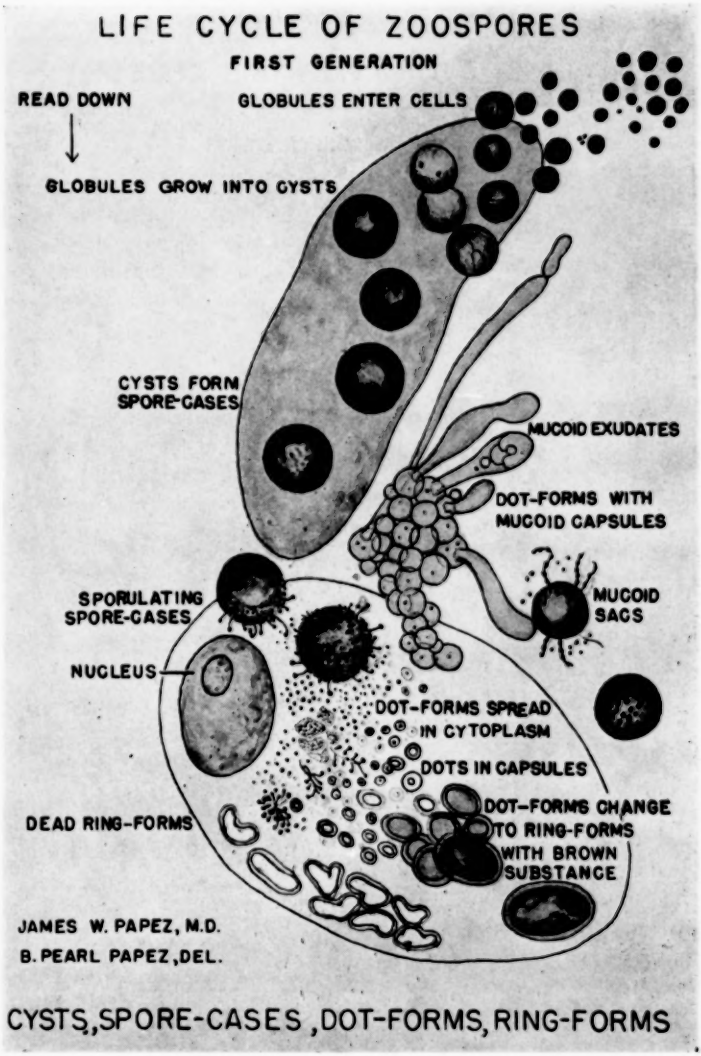

FIG. 1.—Zoospore disease in hypophysial cells in mental illness. At top: purple globular zoospores enter cytoplasm of a cell. There they grow into large spore-cases. Small dark dot-forms or elementary bodies appear in the surface of the spore-case. Below, center: sporulation takes place as dot-forms break through surface of spore-case and enter surrounding cytoplasm. This is the infectious stage of the disease. Dot-forms grow in size, acquire an outer capsule with mucin inside in which they oscillate. Dot-forms become larger and acquire a clear center or nucleus which changes to brown substance. At bottom: many of these ring-forms die, shrivel, and acquire lipofuscin around them.

In center: dot-forms are shown in large capsules containing mucoid substance. When capsules are ruptured, tubes of muciod substance grow out. They pinch off to form spherical mucoid exudates, which collect and shelter small zoospores.

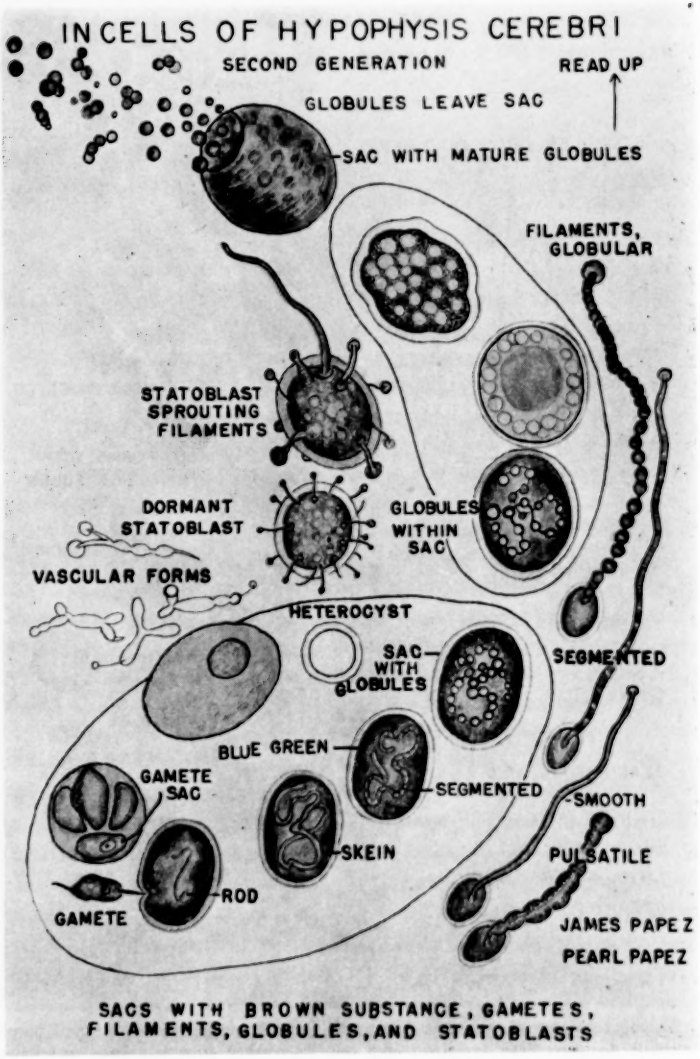

FIG. 2.—Development of ring-form zoospores to form globules or hormogonia. At bottom: sacs with a nucleus or center of brown substance. Within this substance grows a rod, or a skein, which elongates into a folded filament. The smooth filament becomes segmented, and finally globular. The brown substance changes to a blue-green color. When the sacs are ruptured (in process of preparation) the filaments escape, as seen on right side of figure. At top: sacs with mature globules in host cell break naturally and discharge mature globules into tissue. Lower left: gamete cysts with stiff bristle develop in some cases. Empty heterocysts occur. Center: brown substance with globules is discharged early from sacs to form statoblasts. The statoblasts are brown, or blue-green. They have hooks on margins which often give rise to filaments that bear globules at end. Water-vascular forms of several shapes are present.

dropper to suck out some of its cells which were then put into a drop of saline on the microslide. All slide preparations were examined under dark contrast phase, 97 ×, oil immersion microscopes at a magnification of 970 diameters. The record was made at once.

The anterior or distal, secretory part of the hypophysis cerebri was examined in all cases. This part contained many secretory cells; some held together in large groups, while other cells were separate in the saline. The nuclei of these cells and their nucleoli were cloudy. The normal cytoplasm around the nuclei consisted of secretory, silver gray granules, mostly shapeless and joined together. We identified these large cells as basophil cells. There were smaller cells with darker and more amorphous granules, considered as the acidophil cells. Usually there were many nuclei of secretory cells in each of which the cytoplasm was destroyed by the organisms, or was broken away when the preparation was made.

The colonies of zoospores in the cells were usually abundant, and distinctive in appearance (Fig. 3). They left no doubt of their identity, and of their stages of development. These stages varied in different cases. Several stages were readily distinguished after we had gained experience with the main features in many cases.

Four stages of zoospore development were recognized. (a) An early stage (Figs. 1 and 3) was featured by clusters of fine, round, black dots embedded in the cytoplasm. Such dots or spores were sometimes seen coming out of larger pigmented spore-cases. We assumed, from many observations, that this was their origin. We have identified these fine spores as the first generation. In the cytoplasm these small forms increased in size, and became surrounded by a clear space enclosed in a thin capsule. As they increased in size the central dots developed a clear center or nucleus, and their surrounding space and capsule became well defined. (b) A more advanced stage (Figs. 1 and 3) was indicated by heavy ring-forms of medium size. The ring-forms were usually in groups, but often they were scattered in each cell. The cells were widely populated by these organisms. These ring-forms were a characteristic feature of a large number of cases. They gave rise to the second generation. (c) Round sacs or protoplasts of fairly large size contained a nucleus or center of a brown-red and often blue-green secretion of mucoid (pectin-like) substance. In this substance a growth took place which began as a rod, lengthened into a filament that became segmented and finally globular. Mature sacs of this generation had globular zoospores in them (Figs. 2, 4, and 6). Cysts or regular, round spore-cases of purple color grew singly or in groups of two to four cysts in a cell. They were seen frequently in the cytoplasm of pituitary cells. Black

pigment often occurred in them as numerous dots. This indicated a shattering of their inner contents, and ushered in the growth of tiny new forms described above as the first generation. Cysts of several varieties grew out of the spherical forms described above as the globular

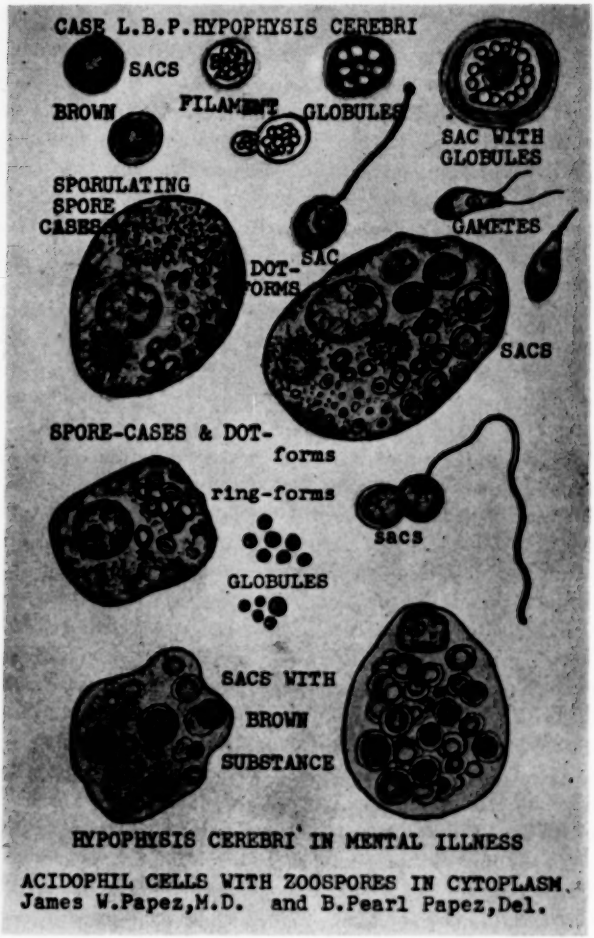

FIG. 3.—Acidophil cells from distal hypophysis cerebri in mental illness. The drawings show globules, sporulating spore-cases in the hypophysial cells; they give rise to dot-forms, growth of dot-forms into ring-forms and sacs, with brown and blue-green substance in their center, in which grow filaments that produce globules or hormogonia, two ruptured sacs are shown with escaped filaments, and two gamete cysts each with eye and bristle. X2000 diameters.

zoospores. The gamete cysts, the pulsatile forms, and statoblasts arose from the nuclear brown substance in some of the sacs. All the above forms were seen in the cytoplasm of hypophysial cells.

Living zoospores in a state of rapid oscillation and Brownian movement, swarmed in the sterile, normal saline. They were the following: (a) Cysts or spore-cases of several varieties were present (Figs. 1 and 3). There were small, round ones of purple color. Those with black spots of pigment were often shattered and seemed to be old ones giving rise to fine dot-like forms (Fig. 3). The angular, purple ones or gametes were vigorous explorers feeling about with a protruding blunt end, or with their flagella (Figs. 3, 4, and 5). (b) Clusters of globular zoospores on a common base were numerous (Figs. 1 and 5). They oscillated in an awkward way. When old, the globules turned into rings with a clear center. We think they came from spore-cases. (c) Single globules or two or more globules in a chain were common. (d) Some rods were seen (Fig. 6). (e) Filaments were encountered in many cases (Figs. 2, 4, 6). The smooth ones seemed to be immature forms coming out of sacs and out of statoblasts. Segmented and globular ones were advanced stages of the smooth ones. They moved in a sinuous manner. When they shattered they gave rise to the globular forms. (f) Pulsatile forms were short, stout filaments that came out of sacs, waved about and pulsated (Fig. 2). They were seen to develop into pulsating globular filaments that consisted of a longitudinal internum and a globular externum, both contracting in a peristaltic manner. (g) Statoblasts of the gray-brown variety, with globules in them and hooks on their outer margin, were fairly frequent (Figs. 2 and 4).

An alternation of generations was apparent in the life cycle of the parasites. It consisted of two stages, each a distinctive one exhibiting certain variations. One began with spore-cases, and was a small spore-bearing stage. The spores grew into ring-forms. The second stage was the nuclear sac stage that gave rise to brown substance in which grew filaments to form globules or hormogonia. From many cases it was possible to reconstruct these two stages.

The first generation began as spore-cases or cysts (Figs. 1 and 3). They came from the large brown-purple globules. These cysts were usually seen embedded in the cytoplasm of the hypophysial cells. They developed granules of dark pigment in their interior. There was a tendency for cysts to enlarge at this stage, and for the granules to protrude from the surface. This stage was followed by release of actively moving dot-like bodies that embedded themselves in the cytoplasm of the cell (Figs. 1, 3, and 4). These elementary bodies represented

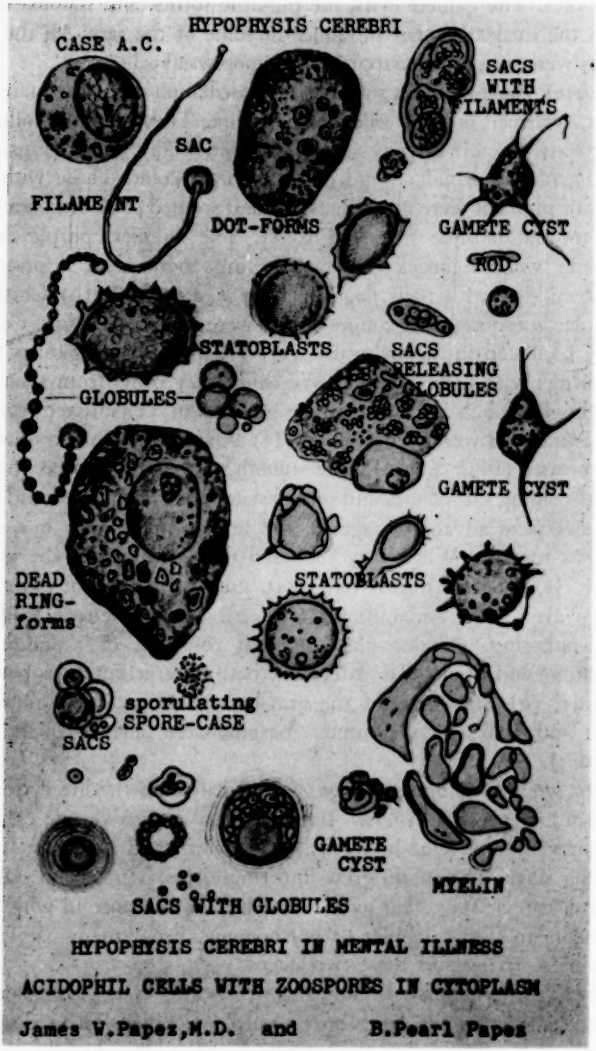

FIG. 4.—Acidophil cells from distal hypophysis cerebri in mental illness. The drawings show small spore-cases sporulating into cytoplasm of upper two cells; this produces small dot-forms which grow into ring-forms, some into large sacs. The center or nucleus of sac give rise to filaments, first smooth then globular, globules in sacs (at bottom), sacs releasing globules (center), gamete cysts which produce exploring gametes with eye and probing bristles, statoblasts, and myelin figures from broken nerve fiber sheaths. About X2000 diameters.

the first generation. Soon they developed a clear ring around them. They grew into large, ring-forms with a clear center and a dark contractile purple ring (Figs. 1 and 3). They were surrounded by a clear capsule containing fluid in which they oscillated (Figs. 1 and 5). Some of these large ring-forms became filled with brown substance

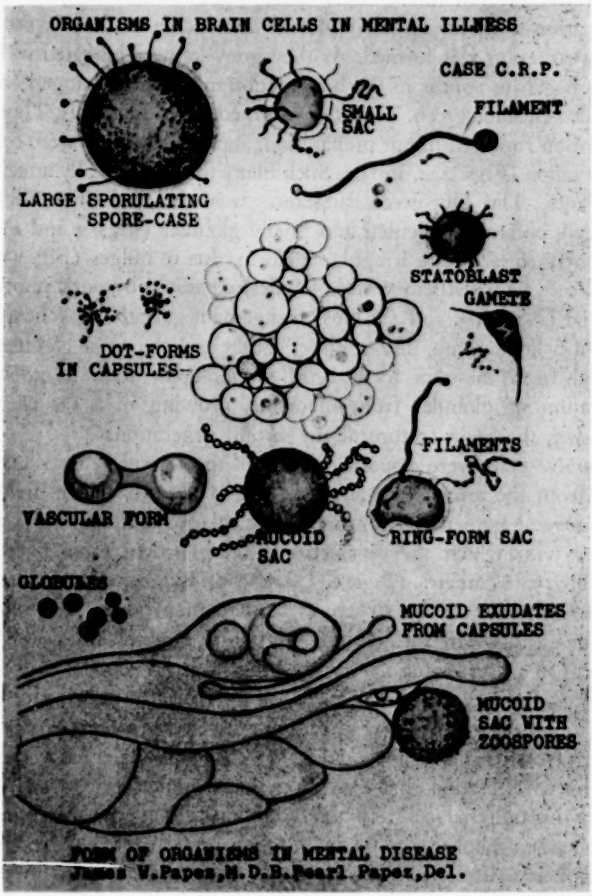

FIG. 5.—Organisms in brain cells and hypophysis cerebri in mental illness. The drawings show some characteristic zoospores, namely, a large sporulating spore-case, below it two small discharged group of dot-forms, growing dot-forms within mucin-filled capsules (center), mucoid exudates from ruptured capsules (bottom), a mucoid sac collecting loose zoospores (center), mucoid sac with collected zoospores inside (lower right), ruptured sacs with brown substance and escaped filaments, gamete with two eyes and two bristles, and a statoblast (upper right). About X2000 diameters.

(probably pectin, plus glycogen, plus phycocyanin). These large ring-forms became the sacs of protoplasts that gave rise to the second generation. A large number of ring-forms seemed to be sterile, or had given rise to statoblasts. These dead ring-forms shriveled and became encased in lipofuscin, forming the typical lipofuscin granules (Fig. 1).

The second generation of zoospores began as sacs (Figs. 2 and 3). They arose from the large ring-forms in which a nucleus or center of brown secretion was formed. As they grew larger, elongated rods and skeins began to appear in the brown substance which changed color to an iridescent blue-green. The skeins formed coiled filaments. These sacs were often ruptured in our preparation, and the filaments were released in the saline (Figs. 2, 4, and 6). Such filaments were usually attached to their sacs. The blue-green substance was absorbed. The filaments enlarged, became segmented and finally globular (Figs. 2 and 4). The large mature sacs were irregular in form, due to bulges (Fig. 4). The round sacs had in them white globules, separated by dark pigmented material (Figs. 2, 3, and 4). This arrangement gave the sacs the appearance of a honeycomb. The single globules and chains of globules that escaped from the sacs formed the second generation of zoospores. Formation of globules from filaments, growing in a sac or protoplast, was the main feature of the second generation.

Gamete cysts were a special feature of the second generation. They came from the brown substance of some of the sacs which divided to form several gametes. They had a central eye and one or two stiff bristles with which they pierced the other brown sacs. They were probably male gametes. (Figs. 2, 3, 4, and 5). Some conjugated.

Disk-forms were flat structures seen in many cases. We supposed that these disks were formed by longitudinal compression of a globular filament in a narrow tubular sac; but this origin was not seen.

Statoblasts were of three kinds. (Fig. 2). The yellow-green ones were round or oval in form and usually flat. They were 5 to 7 microns in diameter, smaller than a red blood corpuscle. They consisted of a firm brown secretion that exuded perhaps prematurely from nuclear sacs in hypophysial cells. In this secretion, globules were embedded. Characteristic hooks, 9 to 13 in number, projected from the margins of statoblasts. In some cases the hooks grew out as stout projections which moved slowly. They gave rise to globular zoospores, usually attached by stems (Fig. 2). The golden statoblasts were formed in the brown substance. They had globules in them, and sharp spines on the outer border. They were the most numerous variety of statoblasts (Fig. 4). The bur-like statoblasts were similar to the preceding, but spherical with spines all over their surface. The statoblasts were prob-

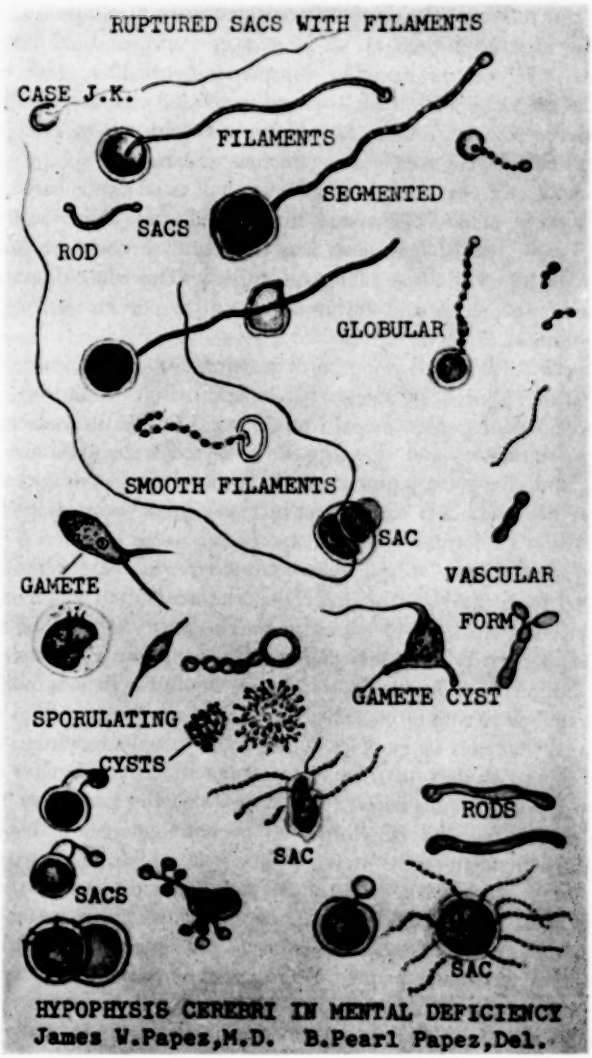

FIG. 6.—Zoospores seen in hypophysis cerebri in a case of mental deficiency. The drawings show ruptured sacs with extruded filaments (above), a single pulsatile form (below center), (below it) two sporulating spore-cases, to each side gametes with bristles, ruptured sacs with brown substance, with extruded filaments, rods, and vascular forms. About X2000 diameters.

ably dormant seeds designed for transportation, attachment to tissue, or some survival purpose not yet clear to us. Parasitized red blood corpuscles were seen in many of our preparations. They gave rise to globules on stems. Statoblasts changed to mucoid sacs (Figs. 1, 5).

Heterocysts were round, clear empty cysts with a firm, double wall (Fig. 2). They were usually 3 to 7 microns in diameter.

Mucoid sacs were clear, round spheres of exudate that arose from statoblasts in some degenerated hypophysial cells (Figs. 1 and 5). Their origin was always clear, and their growth could be followed from day to day in the saline suspensions. The mucoid sacs were a device for gathering and storing exhausted zoospores under unfavorable conditions of survival.

Vegetative structures were parts growing around or outside of the reproductive spores, globules and gametes (Figs. 2 and 5). They formed hoods, capsules, mucoid tubes, etc. The capsules secreted the mucoid substance; and the ring-forms secreted the substance with brown and blue-green pigment in which the filaments and globules as well as the statoblasts were formed. The vegetative forms presented a pink-brown color and slow undulating movement.

The neural part of the hypophysis cerebri or pars nervosa was removed from the gland by dissection. The neural part had elongated pituicytes stretched out in the solution. The nuclei of these cells were either elongated or round. Groups of dead ring-form zoospores were seen within the pituicytes. In some cases lipofuscin deposit was present around these ring-forms. There were also round sacs, each of which contained filaments or globules. Other round sacs had swiftly swarming zoospore clusters in them. We supposed that these were in an activated stage. In the saline, there were swarming great numbers of globular clusters, and ring-forms on a common base. Smooth and segmented filaments were present. They projected from the ruptured sacs. There were present thick, slowly bending rods, and a variety of nimble rods, some smooth and other segmented, with a globule at one end. Cysts or sporecases of purple or blue-green color, with dark pigment in them, and gamete cysts that moved about and explored, were among the living occupants of the neural hypophysis. Such zoospores were also found in the infundibular stalk. In many cases the neural part was occupied chiefly by zoospores of the first generation.

The intermediate part of the hypophysis cerebri was examined separately in several cases. The same type of infestation was present. It seemed to us that the condition of parasitism in the intermediate part was less advanced than that in the anterior part of the gland in the same case.

The hypothalamic floor was removed with infundibulum in several cases. Suspension preparations showed some nerve fibers, and nerve cells with zoospore organisms. The saline contained the same organisms as were found in the cerebral cortex and hypophysis.

The hypophysis cerebri in mentally deficient children under ten years of age showed only the small dot-like organisms and small ring-forms, rods, filaments and cysts (Fig. 6) None of the old forms were present, either as single ring-forms or sacs entombed in lipofuscin. In several cases of deficiency under 20 years of age the large lipofuscin granules were more numerous after the twelfth year and were definite at the age of 18. In young schizophrenic patients, the hypophysial disease was also in its early stages. In the mentally deficient patients there was an extraordinary destruction of brain tissue by the same zoospore disease, often associated with lack of development or maldevelopment of cortex, thalamus, and other parts of the brain.

In patients with psychosis of long standing and repeated attacks with deterioration, the gland was more diseased. Many of these patients were thin and emaciated at the time of autopsy. A larger number of gland cells was affected and the population of the parasites was greater in each cell. Usually the destruction of cytoplasm was also greater; and with this a larger number of cell nuclei without cytoplasm was present. The size of the gland was usually diminished as judged from the amount of space around it in the sella turcica. Some glands were clearly atrophied by loss of cells, but others without atrophy were so full of organisms that the loss of cytoplasm from the cell nuclei did not result in much shrinkage. The part of the cytoplasm of individual cells not occupied by zoospores was as a rule preserved and gave the appearance of normal secretory granules.

In old patients with a psychosis of recent origin without much deterioration, the hypophysis was fairly well preserved, and the disease of its cells did not have the same chronic residue of dead organisms entombed in lipofuscin. More important than age of the patient was the time of onset of the psychosis and its duration.

Comment

The over-all pattern of hypophysial disease in psychoses was a consistent zoospore infestation, similar to what we found in the cells of the cortex and thalamus. Whatever stage was present, there was much variation in severity of cell destruction, and density of organisms. In cell destruction, the secretory cells lost their cytoplasm so that their nuclei were bare. In cell infestation, the cytoplasm was partly or wholly occupied by zoospores in various stages of development.

It was here that stages of entrance and growth were readily observed. They were often represented by dead zoospores that remained as fixed objects in the cytoplasm. The living zoospores seen in the saline had dropped out of the cytoplasm of cells, when the preparations were made. Young, recent, old and dead zoospores were identified in most cases. The young stages were the small dot-forms. The medium-sized organisms were ring-forms. Transitions from the dot-forms to the large ring-forms were often seen. When dead, ring-forms showed as a purple, crinkled externum and a clear internum. Old dead forms were often surrounded by yellow lipofuscin. New crops of organisms in the spore or elementary body stage, often overlapped old stages of dead organisms.

Lipofuscin granules were commonly found in the hypophysial cells. Great numbers of the ring-forms became inert when they reached a large size and were surrounded by fat. Material from children under nine years of age showed living contracting ring-forms of various sizes without lipofuscin. In older children the dead and crinkled zoospores became surrounded by lipofuscin. The desposition of fat was slow, so that stages could be recognized. Old lipofuscin granules were about 12 to 18 years old in terms of the original granule. They occurred also in walls of blood vessels occupied by zoospores, in the sclerotic patch, atheroma and myocardium. The phase microscope showed that dead zoospores and dead sacs formed the central core surrounded by fat. Hitherto, lipofuscin granules have been regarded as a simple form of fatty degeneration. Lipofuscin granules and their stages of development indicated an interval of time in the course of the disease.

Duration and recurrent attacks of mental illness seemed to correspond to stages of zoospore disease in the hypophysis. New crops of zoospores in the dot-stage overlapped older stages of dead ring-forms and sacs. It was this overlapping of older stages by young forms that gave us a rough idea of the duration of the disease and stages of life cycle at time of death. In most cases a long period of growth and repeated dissemination were inferred. From young cases and those with one severe attack near the end of life we estimated that nine to 11 years (more or less) were required for completion of all stages of the life cycle of the zoospores. To this was added a period of dying of old ring-forms with formation of yellow lipofuscin around them.

In the two main parts of the gland, the stages of the disease were often different. In cases of recent illness the pituicytes of the neural part showed an earlier stage of disease. In illnesses of long standing the secretory cells of the distal or anterior part of the gland showed advanced stages of the disease; while the neural part showed a retarded

stage of zoospore development. Did the disease come down from the hypothalamus through the stalk? We examined the infundibulum in a number of cases; it showed the typical zoospore disease. Another reason for retarded development of the zoospore growth in pituicytes was the absence of Nissl substance. In psychoses of recent origin, the neural part was involved first.

In psychoses of long standing there were several crops of zoospores in the secretory cells of the distal (anterior) part. Also their stages were older or more mature. The second generation of sacs with brown-blue-green substance, rods, filaments, and globules in them, was well advanced. The globules, gamete cysts and statoblasts were more numerous, than in the neural part.

The acidophil cells of the distal part of the gland were often densely occupied by zoospores; their cytoplasm was disintegrated. Cytoplasm of the basophil cells was more often intact, and only partly invaded by colonies of zoospores. The massive colonies of dead ring-forms and large sacs entombed in lipofuscin were found chiefly in the basophil cells. Cells of the pars intermedia and pars tuberalis were also affected by the zoospore disease.

The biologic nature of zoospores was determined by their many and varied characteristics. These corresponded closely to those of blue-green algae of Nostoc and Oscillatoria species. They began as globular forms. Globular forms showed transitions to cysts or spore-cases. These were embedded in the cytoplasm of hypophysial cells. The large ones showed dark pigment reminding one of melanin. With this pigment, small dots of spores appeared to come out of the surface of the spore-cases. This sporulation was observed on a large scale in many cases. These dot-forms or elementary bodies were minute, living forms that invaded the cytoplasm of host cells. They gave rise to a new colony of zoospores.

The ring-forms grew from the dot-forms. Around them they developed a capsule in which they oscillated. The clear material in this capsule was probably thin liquid mucin that permitted the movement. Extensive deposits of these mucoid droplets occurred in many brains. The zoospores seemed to be attached to the bottom by one end. As they increased in size they formed a ring whose center remained clear. Some ring-forms gave rise to a new structure which we called a sac or protoplast which began the second generation.

The second generation of zoospores grew out of some large ring-forms. These produced a brown substance in their center. In this grew rods, filaments, globules, gametes and statoblasts. The brown substance increased in amount and became blue-green in color. At

the end of the growth, the large sac was filled with globular forms, surrounded by dark pigment which gave the sac a honeycomb appearance. In many preparations the sacs were broken open so that the rods, filaments, or globules escaped into the saline. They were usually attached by one end to the inside of the sac. We assumed that normally the sacs ruptured and released the mature globules into the tissue. There they entered the hypophysial cells. By repeated dissemination of the globules, and by many generations of the disease it became wide spread through the tissues.

Gamete cysts were produced in the brown substance by some of the ring-forms. The gametes had one or two stiff bristles or flagella, which they used to pierce. Some had a blunt snout with which they explored actively. The gametes were round or triangle in shape with undulating sides. On the inside they contained a white globule related to the flagellum, with a black spot in the globule and pigment around the globule. This gave to the structure the appearance of an eye.

Statoblasts were made of brown substance. They contained globules, with hooks or spines sticking out of the surface. In many instances, just the brown substance without globules was present. In some statoblasts the globules were moving slowly. In the mature, chartreuse-colored statoblasts, the hooks became stout, mobile processes which moved them around. We thought that the statoblast was a dormant seed which remained embedded in the tissue for some time.

Heterocysts were spherical structures with double walls and fluid contents without globules.

Vascular forms contained only fluid. Their thick walls were purple and often undulated. They took various shapes such as a club, test-tube, triradiate tube, series of diminishing spheres, and other shapes. The tubes and rods had an exploring flagellum at one end.

Vegetative parts and substances were meager but definite. They exhibited spontaneous growth. Among these vegetative structures were included the capsules of the dot-forms and ring-forms, the brown substance, the brown, blue, green and dark pigments, hooks, spines, mucoid exudates, and vascular forms. The exudates oozed out of the damaged capsules around dot-forms and ring-forms. These capsules were able to secrete this clear substance in great quantities. The mucoid sacs were a device for collecting free swimming zoospores which attached themselves to the sac, penetrated it, and became small, resting spores on the inside of the sac. This occurred in old suspension preparation. The vegetative parts did not develop beyond the incipient stages attributed to blue-green algae. The zoospores retained simple reproductive structures and a parasitic mode of life.

The colonial nature of the zoospore infestation was in evidence in the hypophysial cells as well as in the nerve cells of the cerebral cortex. It depended on the local sporulation of a spore-case, or a statoblast.

For the disease we described in the hypophysis we had no precedent to follow in our descriptions. In our researches we have not seen mitochondria and secretory droplets, reported by other investigators. We thought the zoospores might account for some of the objects seen by others, such as the Herring bodies, and the secretory granules of the Scharrer's. Rasmussen referred to a great many small masses of colloid, of disputed origin and significance, found throughout nearly all parts of the anterior lobe in an anthropoid ape. We have not been able to identify our zoospore with histoplasma, toxoplasma, or true fungal forms. The zoospores did not belong to any order of bacteria. They resembled organisms of the pleuropneumonia group, and some of the rickettsial types. They resembled some insect viruses.

We have given a complete account, of a specific zoospore disease. This truly remarkable disease of the hypophysis cerebri caused by an infestation with blue-green algae was seen with great clarity through the dark contrast, phase microscope in over 100 cases of mental illness.

Summary

1. One hundred hypophyses from mentally ill patients were examined post-mortem by means of saline suspensions under oil immersion, dark contrast, phase microscopes at magnifications of 970 diameters. Handling and equipment were sterile.

2. Suspensions of distal and neural parts were made by placing a drop of sterile saline on a wide microslide, and gently dipping a cut surface of the gland in the drop to make a turbid suspension. Coverglass was sealed to the slide with Vaseline.

3. Immense numbers of living and dead zoospores were seen in all their variations in the cytoplasm of secretory cells and pituicytes.

4. Two alternate generations of zoospores were found in all parts of the gland in various stages of development.

5. The first generation came from small brown or purple, globular cysts or spore-cases which were lodged in the cytoplasm of the hypophysial cells. The spore-cases became pigmented and sporulated: they gave rise to fine pigmented, dot-forms or elementary bodies, which invaded the surrounding cytoplasm where they formed a colony. The dot-forms were seen as fine, black dots surrounded by a clear space within a thin membranous enclosure.

6. The small dot-forms grew into large ring-forms with a clear

center inside of a purple ring, and an outside enclosure. Many of these ring-forms died and became crinkled lipofuscin granules.

7. The second generation was a development of some large ring-forms into large sacs. The clear center was filled with a thick brown secretion. In this, a filament developed. At first it was short and smooth, then long and segmented and finally it broke up into globules. The brown secretion became blue-green and was used up during this period of growth of filament into globules. It was replaced by dark pigment between the globules. These round bodies escaped from the sac to form swarms of freely moving globules. They were pink-brown or purple in color, and entered the hypophysial cells. In the cells they formed a new crop of pigmented spore-cases that sporulated and gave rise to the dot-forms of the first generation.

8. Some ring-forms gave rise to large, purple, angular gamete cysts with a pigmented eye on their inside and one or two flagella with which they explored vigorously.

9. Statoblasts or resting seeds were composed of the brown substance filled with globules. Hooks or flagella projected from the surface and often gave rise to short, stout, moving filaments. Statoblasts were a resting seed. They gave rise to mucoid socs.

10. Mucoid exudates exuded from opened enclosures in damaged colonies. They attracted and collected wandering zoospores which entered them and became small resting spores. Zoospores had a number of ways of insuring widespread progagation, transport, attachment and survival under various conditions.

11. New crops of zoospores overlapped old ones. Growth was slow, from nine to 13 years, for the two generations. Cell cytoplasm was locally destroyed; what remained was normal.

12. Zoospores appeared to be *blue-green algae* of *Nostoc species.* Dead colonies in ring stage of development were surrounded by fat to form lipofuscin granules.

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VISUAL CHANGES AND AFFECTIVE STATES*

E. I. STRONGIN
AND
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IN COLLABORATION WITH
NINA BULL

In a previous report (6) it was shown by means of ophthalmographic recordings that the expectation of electric shock produced a definite effect on visual functioning in 80 per cent of a group of normal subjects.

The changes recorded were in the direction of impairment of function or improvement of function—these being the aspects of the investigation in which we were particularly interested. Among the functions measured were binocular co-ordination and steadiness, which are of special importance for good visual performance.

Our findings were later interpreted in terms of attitude theory as presented by N. Bull in a series of papers (2, 3, 4) and a monograph called *The Attitude Theory of Emotion* (5). The theory postulates preparatory motor attitude as the basis of feeling, and stresses the orienting function of vision in all affective states.

The present investigation, which is part of a continuing project for the study of motor attitudes in emotion, was undertaken to determine (a) whether a difference could be demonstrated between the visual performance of normal subjects and that of patients coming for psychotherapy; and (b) whether improvement in emotional attitudes as brought about in patients undergoing psychotherapy could be correlated with changes in their visual functioning. Ophthalmographic recordings were made and binocular co-ordination and steadiness were measured.

The normal control group consisted of 20 subjects who were not undergoing psychotherapy and who were functioning in a generally satisfactory manner. They ranged in age from 20 to 40. The data on these subjects were obtained in the years 1940-42 during which time the study of vision in affective states began but was interrupted by World War II. The procedure used in obtaining these data is

* From the Psychiatric Institute, New York: Research Project for the Study of Motor Attitudes in Emotion.

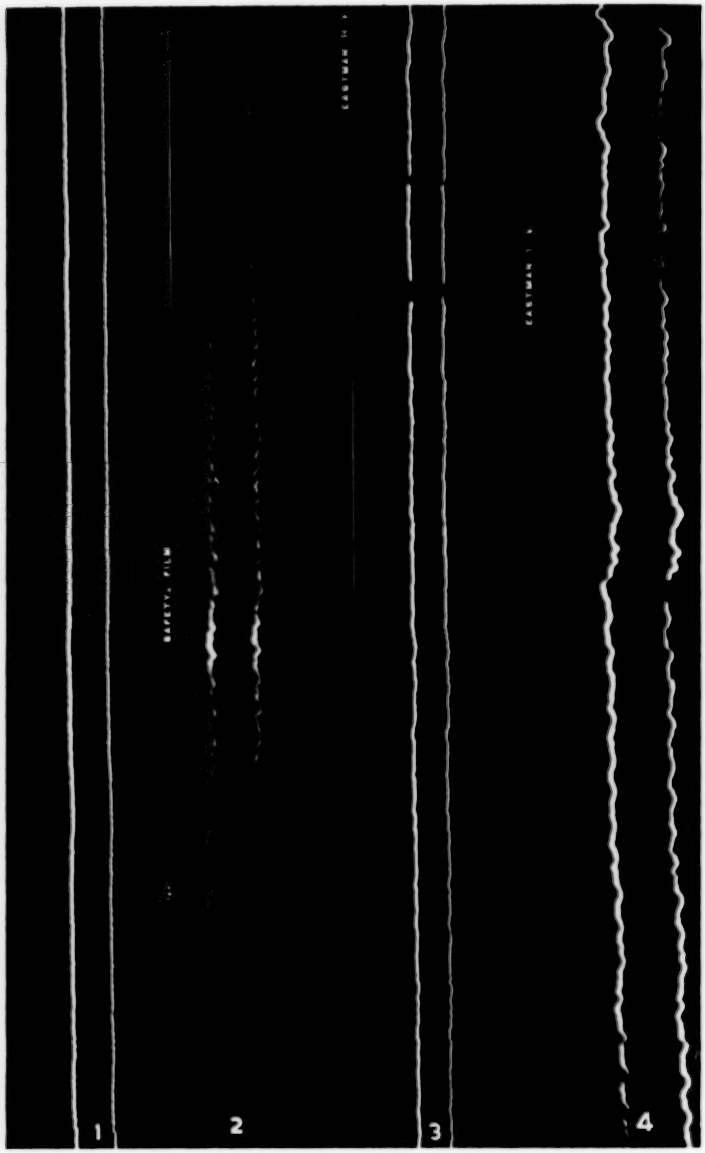

FIG. 1.—Eye movements recorded in normal and neurotic subjects.

1. Normal (control) subject, showing good binocular co-ordination and steadiness.
2. Neurotic patient in emotionally disturbed state showing impairment in binocular co-ordination and steadiness.
3. Same patient one hour later showing pronounced improvement in binocular co-ordination and steadiness which correlated with improved emotional attitude.
4. Neurotic patient with complaints of inadequacy, showing impairment in binocular co-ordination and steadiness, which persisted with little change as did the psychologic symptoms during the period of the experiment.

described below and is the same as that used in the recent study on psychoneurotic subjects to be reported in this paper.

The experimental group consisted of 10 patients who had come to a private office for help with their emotional problems. The first 10 persons who were treated long enough to permit forty tests to be made were selected for inclusion in this study. All of these subjects fit into the nosological category of Psychoneurosis. Within this category they were differentiated further on the basis of their symptoms into three groups as follows:

Group A. Consisted of 4 patients whose main complaints could be classified as psychosomatic. In each case there was one predominant physical symptom which, while not continuous, was recurrent. These patients were medically examined and no organic basis found for their difficulty. The main complaints were as follows: one subject suffered from vomiting, one from dizziness, one from skin eruptions, and one from tachycardia.

Group B. Consisted of 3 patients whose complaints were mainly of inadequacy: (a) They did not work efficiently; (b) they started things and did not finish them; (c) they were full of self-blame for this.

Group C. Consisted of 3 patients, all of whom required help because of a specific incident in their lives which had overwhelmed them. In each case the precipitating situation was indeed severe. However, as treatment progressed it became obvious that other factors were present which had also contributed to the inability of these patients to cope with the impact of the original precipating incident.

Experimental Procedure

The subject was seated at the ophthalmograph and instructed to keep his eyes focused on a black X placed on a blank white card. While the instructions were being carried out, the ophthalmograph recorded on a moving film, for a period of 44 seconds, all the movements made by both eyes. The film was later analyzed in terms of binocular co-ordination and steadiness (1). Binocular co-ordination was measured by the parallelism of the two lines appearing on the photographic record, while steadiness, which we considered as ability to maintain visual focus on a point requested, was measured by the absence of wavering and/or nystagmus. For purposes of short-term comparison, readings were taken at the beginning and again at the end of each psychotherapeutic hour. The number of tests on each subject ranged from a minimum of 40 (done during 20 sessions

over a period of two months) to a maximum of 60 (done during 30 sessions over a period of four months).

Findings on First Problem: (Comparison of Normal with Neurotic Subjects)

1. *Control Subjects.* These were able to maintain fixation for the period required with no appreciable disturbance in binocular co-ordination or steadiness.

2. *Experimental Subjects.* All but one of these showed decided impairment in one or both of the above two functions when first measured.

Since the situation in which the tests were made was not and could not be the same in the two groups, this comparison is suggestive rather than definitive.

Findings on Second Problem: (Comparison of Contrasting Emotional States in the Same Subject)

Group A (*with Psychosomatic Complaints*). Of the 4 experimental patients who made up this group, 3 demonstrated a fairly consistent pattern that was not found in the other groups. The pattern was as follows: The patient would come into the office emotionally disturbed and, when tested on the ophthalmograph, would show an impairment in binocular co-ordination and/or steadiness. At the end of the hour, if the patient felt relieved emotionally (with an associated alleviation of physical symptoms), there would be a dramatic reversal of the findings taken at the beginning of the hour. Binocular co-ordination and/or steadiness were measurably improved. In other words, these visual disturbances were correlated with the patients' emotional state and were reversed when the patients' emotional attitude changed. This improvement was not permanent, however, and was not sustained in cases where the patient came back on another occasion in a disturbed state.

One subject in this group, whose complaint was tachycardia, showed a variation from this pattern. She did not demonstrate an impairment in binocular co-ordination and/or steadiness at the beginning of the hour no matter how emotionally disturbed she was. However, when she was tested repeatedly at eight minute intervals, the impairment would appear after the third or fourth test. Apparently she was able to mobilize herself for the first few tests but could not continue to do so.

Group B (*with Complaints of Inadequacy*). In these 3 subjects

impairment in binocular co-ordination and/or steadiness was found at the start of treatment. They showed little, if any, improvement in emotional symptoms during the three months of the experiment, and the visual picture remained approximately the same throughout.

Group C (with Complaints of an Overwhelming Incident). In these 3 subjects impairment in binocular co-ordination and/or steadiness was found at the start of treatment. While they did not show the dramatic improvement noted in Group A, neither did they show the consistent long-term impairment demonstrated by Group B. Here the evidence pointed to a gradual improvement in their visual functioning during the course of psychotherapy as their situational anxiety was allayed and they gradually began to make an adjustment to the environmental changes which had precipitated their illness.

In line with the attitude theory of emotion the hypothesis is here offered that impairment of visual functioning as reported in our findings constitutes a partial etiologic basis for those feelings of disorientation and resulting inadequacy which are so characteristic of emotional disturbance.

This explanation rests on the proposition that binocular co-ordination and visual steadiness are two of the main factors in the orienting mechanism of the body as a whole, and that impairment in these two areas of oculomotor performance gives rise to feelings of disorientation and inadequacy through absence of a clear-cut sense of direction.

The above concept will be elaborated in a forthcoming article on the mechanism of goal-orientation and the manner of its disruption.

Summary

An experiment was undertaken to determine (1) if binocular co-ordination and visual steadiness as measured by the ophthalmograph are the same in normal and psychoneurotic subjects; and (2) if changes in these two functions were related to emotional changes which took place during psychotherapy.

The following three types of comparison were made: (a) the records of normal subjects were compared with those of psychoneurotic patients; (b) the records of patients taken at the beginning of each interview were compared with those taken at the end of the interview; (c) the records of patients taken at the beginning of treatment were compared with those taken after a period of from two to four months of therapy.

It was found that psychoneurotic patients had more disturbance in the functions mentioned above than the normal subjects who formed the control group. It was also found that these visual dis-

turbances could be readily correlated with the conscious emotional state in 9 out of the 10 patients.

The attitude theory of emotion was proposed as an explanation of this correlation, with the oculomotor disturbances contributing to the feelings of disorientation and inadequacy experienced by the patients.

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THE MANIC STATE AS AN EMERGENCY DEFENSE REACTION

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I

Our observations of manic behavior have centered on understanding its place in the continuity of the patient's psychic life. This report is based on psychoanalytic study of almost two dozen patients in manic phases. In making these observation we found that our formulations differed in certain respects from classical views regarding both the symptomatology and psychogenesis of the manic state.

The present concept of the manic syndrome began, according to Lewin, with Mendel's application of the term *hypomania* to a state of hyperactivity and elation without delusions. The manic-depressive psychosis as an entity was introduced by Kraepelin, according to whom mania and melancholia are both manifestations of a single disease process, a disorder of affect, characterized by attacks of elation or depression and not resulting in deterioration. Kraepelin listed the cardinal symptoms of the manic phase as (a) elation, (b) flight of ideas, and (c) hyperactivity. This basic picture was retained by Bleuler and by subsequent standard texts of psychiatry.

Psychoanalysis shifted interest from symptomatology to a deeper concern with the genetic and dynamic problems of the psychoses. Nevertheless, psychoanalytic writings have essentially accepted the conventional sterotyped description of the manic state.

The fundamental analytic observations were those of Abraham and Freud. Abraham drew up the following set of etiologic postulates for the manic-depressive psychosis:

1. Oral predisposition
2. Disappointment in love during infancy before the Oedipus complex was resolved

* This paper was completed shortly before Dr. Bateman's death. We wish to express in this note our appreciation for the stimulating encouragement and helpfulness which Dr. Bateman displayed in his work with his colleagues and collaborators. T.A., B.K., M.W.M., Columbus State Hospital.

3. Repetition of this disappointment later in life, resulting in an oral regression.

Abraham and Freud also pointed out that there are similarities between melancholia and obsessional neurosis, that the patient is not really well during the interval between attacks, that self-reproaches in melancholia represent unconscious reproaches against the lost, ambivalently loved person, and that a cannibalistic oral regression is indicated by fantasies of devouring and necrophagia.

Freud explained the replacement of depression by elation through the hypothesis of a fusion between ego and super-ego, with liberation of the ego from the pressure of the super-ego. The energy previously engaged in the struggle between ego and super-ego is thereby freed and placed at the disposal of the ego, which indulges in a triumphant celebration.

Helene Deutsch and Lewin emphasized the importance of defense mechanisms in mania, especially denial. Deutsch also recognized the aggressiveness of manics and the defenses which they employ against it. Almasy and Schilder indicated the defensive character of the manic's activity, his preoccupation with a "painful past" and the fact that elation or happiness is not always present. Lewin, assuming that in the manic state the elated mood is central, suggested that the "happy mood" of the manic is a subjectively valid repetition of an "element in the nursing situation."

In general, the psychoanalytic literature has indicated that in mania the essential problem is a great ambivalence toward an orally incorporated love object. The actual manic attack is supposed to correspond to an escape of the ego from super-ego pressure (guilt feeling) creating a state of elation with exaggerated self-esteem. In this state, however, the defensive mechanisms, such as denial, can be easily recognized.

II

The phenomena of the manic syndrome are familiar yet not easy to characterize with precision. We notice a proneness to talk and act with a minimum of deliberation and with a progressive momentum (pressure). The listener becomes fatigued by the constant diversions and shifts in the sequence of thought, which is complicated by diffuse detail and yet completely restricted to a narrow range of personal references and preoccupations. Inevitably the patient reveals an unabashedly self-centered, unadaptive, aggressive, and superior attitude, even though he sometimes makes an attempt to be friendly and co-operative. He may be smilingly talkative and ready with witty comment or

retort, yet he is basically unhappy and ready to become bitterly sarcastic, impulsively vulgar, or obscene. His brightness shifts to open hostility the moment he feels frustrated, threatened, or belittled. He is shrewdly perceptive, but tense, changeable, and thin-skinned. The manic is never truly carefree, relaxed or at peace, and his laughter is never comfortable or happy. The patient is under such pressure that even when aware of his exhausting, inconsiderate behavior, and actually apologetic for it, he still cannot stop himself.

III

The psychologic significance of this disorder becomes clearer when attacks are related to the life course. It is our observation that mania represents a specific *post-traumatic* stage in the mental life of the patient. This phase can be characterized by its source, its aim, its dynamism, and certain specific mechanisms.

Source. A survey of manic life histories from the psychoanalytic point of view discloses a typical pathogenetic sequence. After an especially traumatic childhood, the patient, who becomes arrested at the infantile level of the pleasure ego, develops an obsessive-compulsive character with a highly unstable equilibrium. Furthermore, fear of the aggressive cruelty in his personality compels the patient to crave continually and insatiably for additional evidence of approval by others. His mental equilibrium depends largely upon whether or not the world loves him enough. Later trauma can readily overthrow this unstable type of equilibrium.

In our manics' life stories we have noted the development of traumatic situations such as the following:

a. The patient's sadistic cruelty drives his family to break off relations with him or his associates to turn against him.
b. In spite of his carefully observed rituals, the patient is exposed to painful experiences which undermine his shaky self-esteem and result in tempestuous anxiety.
c. Ambivalence toward his relatives makes their actual deaths a serious source of trauma. An unconscious delusion usually develops that the relatives died "against the patient."

To sum up: the trauma is a situation or event which the patient interprets as an act of hatred, although unconsciously he feels it to be a justified punishment or retaliation for his own hatred. The traumatic event precipitates a manic *prodrome,* a shock-like reaction to the trauma in the form of a period of perplexity or paralyzing tension and helplessness, signifying the collapse of the patient's compulsive neurotic

defenses in the face of his aggression and guilt. The full-fledged manic psychotic picture then develops as a mobilization of multiple primitive ego defenses to cope with the psychic emergency precipitated by the traumatic life situation.

Aim. The manic state exhibits a primary and secondary aim. The primary aim is emergency restoration of psychic equilibrium in the face of the trauma and abreaction of the traumatic life experience. The secondary aim is protection of the personality from interference by others, while engaged in carrying out the primary aim. Essentially, the manic is striving to master the traumatic situation.

Dynamism. The prepsychotic neurotic defenses proved inadequate to cope with the tension generated by the traumatic experience. In this predicament the manic psychotic dynamism is manifested as the energetically speeded up and consequently indiscriminate, simultaneous employment of multiple mental mechanisms. The patient resorts in particular to the "first aid" mechanisms which characterize the early defenses of the immature ego against objective pain and objective danger. The manic's productions accordingly disclose an attempt to prove, all at one time, that he is innocent in the traumatic situation, that others are guilty of causing his trouble, that he is unaffected by the trauma and that actually no trauma took place. The manic technique is illustrated by an anecdote which tells of a woman who failed to return a borrowed pot. She excused herself with the statement, "I never borrowed the pot, furthermore the pot was broken when I borrowed it, and besides I returned it long ago." In this undiscriminating psychologic opportunism we see how the manic ego can tolerate no delay in its need to eliminate the distress evoked by the trauma.

Mechanisms. The "first aid" defenses characteristic of the manic psychosis are actually common phenomena in childhood (stage of the pleasure-ego). These mechanisms were described by Anna Freud as the "preliminary stages of defense" and listed as follows: denial in word and act, denial in fantasy, identification with the aggressor, restriction of the ego, and altruistic surrender. Considered normal and necessary during the immaturity and dependence of childhood, they mark a break with reality when employed in this fashion by the adult.

IV

Correlating our psychogenetic and dynamic data, we perceive a fuller range of meaning in the manic's symptomatology. We see a deeply disturbed person, strongly attached to life and to people—attached, however, not by love but by feelings of hate and guilt. When this type of basic attachment is reconfirmed by a recent traumatic experience, the

patient's earliest unconscious feelings of disappointment are given new momentum. This increases the sense of his own frustration and results in destructiveness leading to additional feelings of guilt. These guilt feelings can be evaded and the consequent tension relieved only by desperate vigilance and by a psychologic counteroffensive which is no longer consistent with reality. This is the basic meaning of the manic reaction. It is not sufficient for the manic merely to ignore painful recollections or stimuli. He must actively evade, repudiate, or counter-attack them. Therefore, the manic mood is one of extreme tenseness. The patient is constantly on guard against disturbing interference and is ready to show active aggressiveness whenever it threatens. It is this aggressive defense which gives the impression of exaggerated self-esteem expressed through extravagant ambitions, demands, boasting and forwardness.

The apparent "elation" which is so often emphasized does not prove to be genuine. The aggressive remarks and actions may often have a comical or humorous effect, and this is especially the case when the aggression is directed against a third person and accompanied by smiling. The more successful the manic's aggressiveness, the more he smiles, in accordance with the basic dynamism of laughter and wit. The patient's keen observation and relentless frankness also provoke amusement in observers. The patient may with apparent objectivity make a meticulously accurate presentation of an absolutely irrelevant detail in order to evade the painful reality of a situation (pseudo-honesty). He may with a characteristic manic twist make a joke out of outwitting someone who disappointed him, and so on. The manic's behavior and speech vary understandably with the circumstances to which he is exposed. Thus sarcasm, cruelty, hostility, superiority, vulgarity and anal terms may all serve when necessary as aggressive defenses.

A further characteristic defense is the flight of ideas. This is the product of a constant compulsion to introduce new details as evidence proving the patient's innocence. These details are of obsessive importance because ultimately they all serve to deny guilt. Although these unessential and evasive minutiae are not only trivial but usually incorrect, the listener is frequently exhorted to check them with alleged witnesses. The manic's pressure of speech then, with its apparently exhausting, aimless and exasperating flow of detail, proves to be aimed directly and primarily at diminishing his own tension. At the same time, a very small number of topics which are disproportionately important to the patient recur with surprising stubbornness. They are complicated by ramifications, however, to such an extent that the feeling of guilt is lost sight of in the welter of detail. Flight of ideas accordingly reveals in

the most striking fashion the manic's compulsive need to abreact a trauma without experiencing the painful guilt feelings which are connected with it.

The manic's activities similarly serve to conceal guilt. As long as he completely dominates the situation, the patient appears in comparatively good humor and relatively free of tension. Hostility appears whenever his activities are interfered with and he feels that he is threatened, unloved, and unsuccessful. The behavior observed can be divided into three categories:

(a) Signs of general tenseness
(b) Repetitive compulsive actions
(c) Flight of activities—parallel in significance to flight of ideas

V.—Rorschach Observations

Wherever possible, the patients in this study were given the Rorschach psychodiagnostic test. Manic patients use many devices to avoid responding: (1) they merely continue talking and ignore the card; (2) they give ludicrous responses; (3) they hurl the cards aside; and (4) they profess not to understand what is required and refer to the blots as "splotches" or "blobs" of ink. The efforts of these patients to dominate the situation, to direct and monopolize the conversation, and to avoid any type of self-revelation are most obvious in a Rorschach setting. For example, a 43-year-old woman, who had been in a manic phase for many weeks, laughed uproariously when handed Card I, but immediately thereafter began to weep. Conversation which seemed directed toward the cards was as follows: "Would it be my pelvis—an x-ray of it? It's twins and quads and a scar on the right—and four blobs of color and it's phony. This is the index finger. A bunch of butterflies. Two crooks in white coats." After throwing the cards on her bed the patient became extremely hostile toward the examiner and it was not possible to secure an adequate record until she had received electric shock treatment.

As the intensity of the manic phase subsides these patients become more compliant, less hostile, and usually give adequate Rorschach records. Our general observations were all made on the basis of 15 protocols obtained from 9 different patients. The number of Rorschach responses of the manic is usually in sharp contrast to his otherwise enormous verbal output. Our response totals ranged from 13 to 67 (median = 19), with the higher response records reflecting consistently more obsessive-compulsive features (much S, high Ddr, M- in Ddr, use of tiny projections, frequent combinations and exhaustive card descriptions).

In contrast, the lower response records emphasize whole and large D percepts at the expense of Dd percepts. (A particularly interesting finding, since manics in their conversation appear to be preoccupied with small details). This could indicate that the manic is not really concerned with details, but uses them only to ward off the threatened return of traumatic material. The tendency toward W and D percepts likewise tends to produce a better than average number of popular responses and also responses which are generally adequate with regard to form quality (F+% usually above 60). One is thus impressed with the fact that manics, though psychotic, are still able to perceive external reality correctly and (at least in this limited sense) show adequate ego strength. In this respect they obviously differ from schizophrenics in the Rorschach situation, since the latter are usually unable to view the cards conventionally.

It is in the areas of emotional lability and emotional regression that manic patients differ most from normal individuals and from paranoid types. We assume that the weighted total of FC values should exceed the weighted total of C and CF values, if the individual is emotionally stable. In our 15 records there are 10 in which the reverse of this occurred. In 13 of the 15 records sum C also outweighs M, this being explained by the great excess above average of C and CF responses. It is important to note, however, that in every manic record there were also FC responses, which probably represents the ever-present capacity of the manic to return to a state of emotional consonance with the external world.

With respect to dysphoria or depressive trends we find that manics also depart from the normal. If we recall that the weighted Y sum in normal records seldom exceeds 1.5, we can readily see that the manic is over-valent to underlying depressive trends. There were nine of the fifteen records which had Y sums exceeding 1.5 (one record being 9.0).

The intellectual drive of the manic patient which can be observed clinically is also reflected in the Rorschach. This can be noted in the consistently high W per cent, the adequate number of M responses, the high F+ per cent, and in the wide content range, word choice, and richness of associative material.

An important Rorschach sign of the manic patient's beginning remission is his reduced preoccupation with frank sexual and anal content (though this applies more to the female patient than to the male). There is a decided tendency for responses to be phrased in a much less "objectionable" way as remission progresses.

We may therefore cite the following general trends noted in fifteen scorable Rorschach records given by 9 manic patients. These protocols

were obtained during phases of the illness other than the acute manic state. In the acute stages patients use almost any device, including physical assault, to avoid the test. In the fifteen records the response range was from 13 to 67, with a median of 19 responses. In general, the high response records showed many obsessive-compulsive features. Outstanding Rorschach trends were the following (a) greater than average emphasis upon whole percepts and large details at the expense of small details; (b) adequate regard for external reality (or perceptual accuracy); (c) ability to view the cards conventionally (sufficient popular responses); (d) emotional instability (much C and CF), but evidence of remaining capacity for emotional consonance with the external world (sufficient FC); (e) strong evidence of dysphoric and depressive trends; (f) considerable "intellectual drive"; (g) wide content range and richness of associative material; and (h) frank sexual content, which becomes greatly reduced as the patient nears remission. As a general rule, the Rorschach findings in this study seem to be in agreement with the clinical observations of manic psychosis on which this paper is based.

Summary

1. Analytical observation of manics reveals no true elation and no random thinking.

2. Our observations further suggest that the attacks are related to past and present traumatic experiences.

3. The manic state can be viewed as an emergency defense reaction against unconscious guilt feelings following a psychologic trauma.

4. The essential aim of mania is to master or abreact the traumatic experience.

5. The dynamism for emergency abreaction of the trauma is characterized by the simultaneous and inconsistent use of immature ego defense mechanisms.

6. The classical symptoms arise through these characteristic methods of abreactions. For example: (a) flight of ideas and activities—a mobilization in words and acts of every conceivable type of self-justification; (b) aggressiveness—a defense against any threatened increase in tension or guilt feeling.

7. All the symptoms, in addition to serving the essential manic aim, also serve a secondary aim of protecting the patient from external interference while he is engaged in his attempt at regaining psychic equilibrium.

8. According to our observations, the manic never really achieves a fusion between super-ego and ego, and never genuinely relives the

nursing situation. These concepts describe a sort of promised land which the manic seeks but never actually attains.

9. The conception of mania as a primary disorder of affect is not dynamically justified. It seems to us that we can more usefully describe it as an emergency effort to regain equilibrium following a psychological trauma.

10. Rorschach findings can be interpreted as congruent with the clinical data and interpretations described above.

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LUNG ABSCESS FOLLOWING ELECTROSHOCK THERAPY*

JACK M. MARTT, M.D.
AND
GEORGE A. SPIKES, M.D.

Lung abscess is apparently a rare complication of electroconvulsive therapy in the United States because only a few mention its occurrence in the American literature. Altschule (2) reported one case of lung abscess occurring in five years of electroconvulsive therapy on 200 patients. Lowinger and Huddleson (8) described briefly a patient who developed a lung abscess one month after completion of a course of electroconvulsive therapy. Ebaugh (3) reported a death from "acute respiratory infection and septicemia" three months after the patient's last shock treatment.

In contrast, the incidence in the foreign literature varies from 0.5 per cent to 2.5 per cent and referral is made to an astounding incidence of 15 per cent in Arabs (12). Roubier (12) encountered a lung abscess developing after the fourth seizure, which healed over a five-week period with antibiotic therapy. Trinch (14) reported 13 cases in some 13,000 patients treated with metrazol shock therapy. Pons Balmes (11) observed 3 instances of pulmonary abscess occurring during electroconvulsive therapy, 2 of which ended fatally. The same author also described 4 fatal cases of lung abscess due to metrazol therapy.

That this complication may occur, and indeed even be fatal, is portrayed by the following case report.

Case Report

F. S., a white male bookkeeper and musician, aged 59 years, entered the hospital on Apr. 10, 1952, for repair of a retinal detachment of the left eye. The patient had been admitted previously to the eye service of this hospital for cataract removal and subsequent enucleation of the right eye. He was a known alcoholic, having been committed to the State Hospital in 1933 with a psychosis of an unknown type. In 1947 the patient required hospitalization at another psychiatric hospital, where he received an unknown number of electroshock treatments with improvement. Allegedly a diagnosis was made at the time of "dementia praecox."

The arterial pressures were 132 mm. Hg and 86 mm. Hg diastolic; the cardiac rate was 86 beats per minute, and the body temperature was

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98° F. He was oriented and co-operative. The right eye was missing, with a satisfactory prosthesis in place. Detachment of the retina in the superior temporal region of the left eye was noted. The mouth was ill-kept with many carious snags. The chest was emphysematous and the thoracic movements were poor. The lungs were clear and the heart was within normal limits. The state of nutrition was fair, and laboratory studies on admission, including hemoglobin, erythrocyte count, leukocyte count, urinalysis, an electrocardiogram, and roentgenogram of the chest, were within normal limits.

Retinal reattachment was attempted on the fifth hospital day and again five weeks later. Following the last attempt, the patient became restless, refused to eat, developed fear of blindness, despondency, and finally was transferred to the psychiatric service, where a diagnosis of involutional melancholia was made. On June 27, 1952, he was given an electroshock which resulted in a petit mal reaction. The patient showed a good clinical effect and ate well for the first time in three weeks. He relapsed after 24 hours and on June 30, 1952, after 0.5 Gm. of intravenous pentothal, was again given electroconvulsive therapy. Four applications were made, but only petit mal type of responses resulted. Following the fourth application, the patient became cyanotic and apneic. Coramine was given intravenously, with return of labored respirations. One hour later the arterial pressures were 70 mm. Hg systolic and 40 mm. Hg. diastolic. Glucose, 5 per cent solution, and 500 cc. whole blood were given intravenously, with restoration of the arterial pressures to 120 mm. Hg systolic and 70 mm. Hg diastolic. The patient complained of severe pain in the right lower chest and the right upper quadrant of the abdomen following this episode. Roentgenogram of the chest several hours later revealed extensive infiltrate throughout both lung fields and a large cavity was noted in the right perihilar region (Fig. 2). The body temperature rose rapidly to 101.6° F. and pulse to 104 beats per minute. An electrocardiogram revealed prolonged QRS interval, right deviation of electrical axis, and defective conduction through right bundle. After cultures of the blood, sputum, and urine were obtained, the patient was given penicillin, 200,000 units intramuscularly every six hours, and aureomycin, 500 mg. intravenously every 12 hours. The following day he appeared worse, with increasing pain in the right chest, cyanosis, and dyspnea, and was transferred to the medical service.

The arterial pressures were 115/85, pulse rate was 110 beats per minute, respiratory rate was 30 per minute, and body temperature 102° F. The patient was acutely ill, moaning, and unco-operative. Dullness to percussion was present over the lower left chest posteriorly and laterally with coarse, moist râles throughout the left lower lobe. The right chest laterally was tender to percussion, with decreased breath sounds and coarse râles heard over the right middle and lower lobes. Pulmonary emphysema was prominent, with inability to cough productively. The heart was not enlarged, was regular, and rubs or murmurs were not noted. The abdomen was flat and there was right upper quadrant tenderness with slight voluntary muscle

guarding. Peristalsis was normal. The blood culture was negative. Acid-fast bacilli were not found in the sputa. Three sputa cultures revealed *Staphylococcus albus,* Corynebacteria, and Micrococcus, and on July 3, 1952, a coliform bacillus. The leukocyte count was 10,800 to 17,800, with

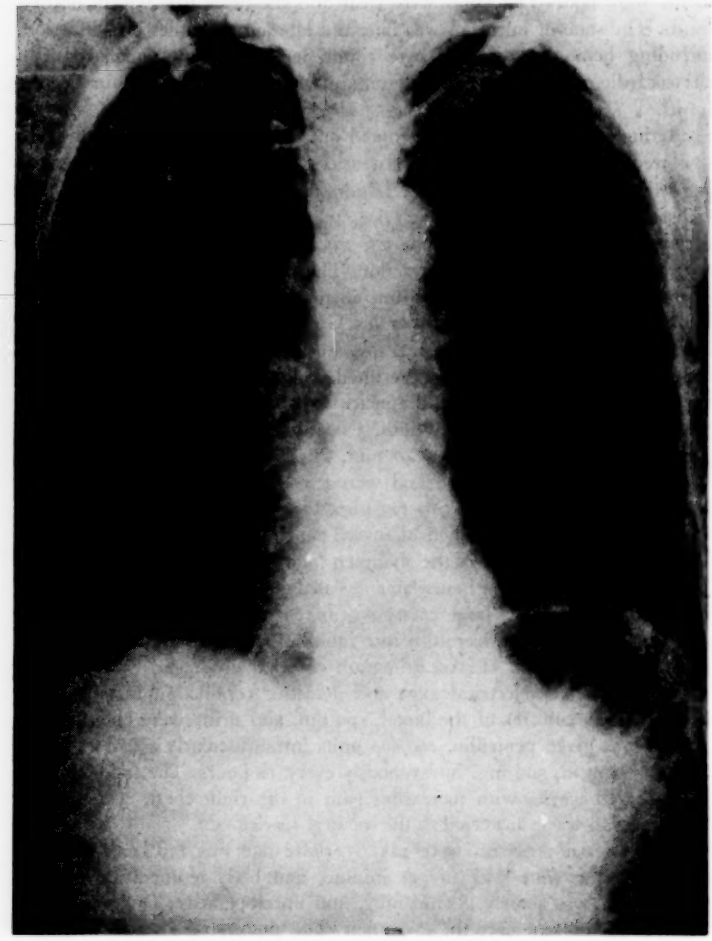

FIG. 1.—PA chest x-ray five days prior to electroshock therapy.

22 stabs, 58 neutrophils, 14 lymphocytes, and 6 monocytes. The CO_2 was 28 milliequivalents per liter, the fasting blood sugar was 116 mg. per cent, the BUN was 24 mg. per cent, the serum amylase was 20 units, the serum sodium was 145 milliequivalents per liter, and the potassium was 4.2 milliequivalents per liter.

Penicillin, 1,000,000 units, was administered intramuscularly every six hours and 500 mg. of aureomycin were given intravenously every 12 hours. In addition, fluids were administered orally and parenterally to 3,000 cc. minimum daily. Nasal oxygen was instituted and tracheal aspiration by catheter was performed frequently. On the third day of the illness, an aspi-

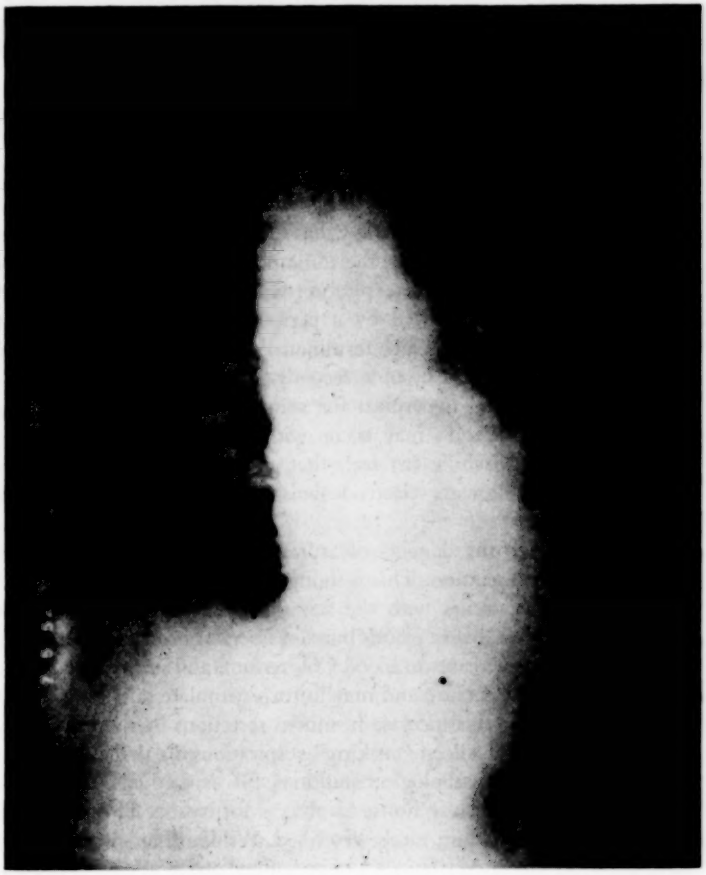

FIG. 2.—AP chest x-ray five days after electroshock. Note abscesses of right upper and lower lobes and diffuse bronchopneumonia of left lung.

ration bronchoscopy obtained a large amount of thick, foul-smelling secretion. Culture of this material revealed a coliform bacillus and *Staphylococcus albus*. On the fifth day the temperature reached 103.2° F. and streptomycin was administered intramuscularly, 1 Gm. b.i.d., and the aureomycin was discontinued. Pain and tenderness persisted over the right

lateral chest and the patient became more dyspneic and cyanotic. On the sixth day a second bronchoscopy obtained a large amount of thick exudate. However, the patient went steadily downhill and, despite vigorous supportive measures, the arterial pressures were suddenly unobtainable on the ninth day. An emergency tracheotomy was performed because of labored respirations and, simultaneously, 1,000 cc. blood was administered intra-arterially. The blood pressure reached 78 mm. Hg systolic, and nine hours later the patient had a small hemorrhage of approximately 30 cc. blood from the tracheotomy site and expired in approximately 15 minutes.

An autopsy revealed "emphysema of the lungs," "pulmonary abscesses, bilateral," and "bronchitis with bilateral bronchopneumonia."

Discussion

The alteration in respiratory mechanics during and immediately following electroconvulsive therapy has been described by Altschule et al. (1, 2). With the onset of the tonic phase of the seizure, there is produced a maximum forced expiration with extreme elevation of the diaphragms. This is followed by a period of apnea which lasts an average of 66 seconds and which terminates with gasping and inefficient breathing for an average of nine seconds. There are then seen maximum inspirations and hyperpnea for some minutes and it is during this phase that aspiration may occur with resultant occlusion of the smaller bronchi. Altschule (2) feels that patchy atelectasis is probably not uncommon following electroconvulsive therapy but that it goes unrecognized.

Contributing to the dangers of aspiration is the profuse salivation which is seen with seizures. This is thought to result from the increased vagal tone which occurs with the seizure. This vagotonic effect not only stimulates the salivary glands but also liberates bronchial secretions. It is also felt that increases in blood CO_2 tension and in the blood lactic acid accompany the seizure and may in turn stimulate salivation. It can be readily seen that this increase in mucus secretions by the respiratory passages, followed by deep "sucking" respirations in the unconscious patient, offers ideal pathologic conditions for aspiration, resulting in atelectasis, respiratory infections, or abscess formation. However, pulmonary complications are rarely described. Williams and Barrera (16) have noted an increased frequency of prolonged apnea as well as respiratory complications in patients who have subconvulsive or abortive responses to electroconvulsive therapy. This is borne out by the case reported inasmuch as only abortive responses were obtained from the shock.

Medlicott (9) feels that respiratory and orthopedic complications are more frequent following "metrazol"-induced seizures than electroconvulsive therapy; however, Roubier (12) reports the incidence of lung

abscess to be statistically the same in both types of induced shock. The latter author also points out that lung infections rarely complicate the first few shock treatments and, in susceptible individuals, an average of eight electroshocks are usually given before lung abscess appears. This, he feels, explains the rarity of pulmonary complications in idiopathic epilepsy since attacks are usually isolated. To bear this out, he also mentions three instances of lung abscess complicating status epilepticus. The onset of lung abscess following electroconvulsive therapy is usually abrupt, with fever, cough, pleuritic pain, and occasionally bloody sputum. However, there may be an insidious course with the abscess not manifest for a matter of weeks (1, 11, 12). The right lung is more commonly affected than the left (12).

It is felt that the most important predisposing factor to this complication is the state of oral hygiene. Food particles in the mouth, carious teeth, and infections of the gingivae and tonsils, all offer good sources for contamination of the bronchial tree. For this reason, dental clearance is an important factor for patients who are to receive electroconvulsive therapy. The use of oral antiseptics and brushing of the teeth prior to each treatment are also strongly urged (11, 12) as a routine prophylactic measure. It is also recommended that a thin, rather than a thick, mouth gag be used for the seizure in order to allow more normal swallowing of saliva (12). Sprague and Taylor (13) feel that prompt and vigorous treatment of the prolonged apneic phases which sometimes follow the convulsions is the major preventive measure of this complication.

In a recent summary of the contraindications to electroconvulsive therapy (16), the following have been listed: (a) recent myocardial infarction, (b) congestive failure, (c) recent cerebrovascular accident, (d) active inflammatory peripheral vascular disease, (e) aneurysm of the aorta, and (f) acute respiratory infections. It is interesting that pulmonary tuberculosis is no longer considered an absolute contraindication (13) and neither is pregnancy nor recent fractures. To the above list, however, we feel that there should be added that group of patients who have grossly infected, carious teeth, and that, until adequately corrected, oral infections in general should preclude electroconvulsive therapy.

Electroconvulsive therapy is, for the most part, still a safe procedure as has been long recognized, and in large series the fatality rates vary from 0.12 per cent (6) to 0.50 per cent (7). Will et al. (15) collected 33 fatalities reported up to 1946. We have found additional reports of eight deaths from 1946 until the present (4, 5, 10, 13, 17). The fact that none of these deaths was associated with pyogenic lung

abscess speaks either for the rarity of this complication or the failure to recognize and record it.

Summary

1. A fatality, complicating electroconvulsive therapy and due to extensive aspiration bronchopneumonia with multiple lung abscesses, is reported.

2. The mechanics of respiratory function following electroconvulsive therapy are reviewed.

3. Poor oral hygience and infected, carious teeth are felt to predispose to respiratory complications and particularly lung abscess, in this type of therapy.

4. Survey of patients prior to electroconvulsive therapy should include dental clearance, in addition to other routine pre-shock studies.

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SOCIETY PROCEEDINGS

MASSACHUSETTS SOCIETY FOR RESEARCH IN PSYCHIATRY

Meeting of November 4, 1953

MILTON GREENBLATT, M.D., President, *Presiding*

SOME THEORETIC CONCEPTS OF THE GROUP PROCESS. JAMES MANN, M.D.

The concepts proposed are born out of repeated observation and experience with a variety of adult groups ranging from psychotic to "normal," male groups, female groups, and mixed groups. "Group therapy is a method of psychotherapy in which the *emotional* reactions of members of the group *to each other* and *to the leader* are understood as being reflections of interpersonal conflicts of the individuals comprising the group. The collection of individuals into a group provides a setting in which these conflicts are intensified and even exaggerated because of the number and variety of pressures exerted by the presence of a group of people. The leader of the group exploits the setting and the emotional reactions for the *direct* general benefit of the *group as a whole,* indirectly for the individual members of the group."

When a group of people is brought together for any purpose, the degree to which the anticipated goals are successfully realized is dependent upon the capacity of the members for genuinely mature relationships. Each group member tends to react with hostility to the anxiety induced by the special setting of group therapy. The working through of the defensive hostility is a necessary preliminary before the leader or group can go on to a consideration of the problems which brought the group together. The ultimate aim in group therapy is one wherein the members are willing to sacrifice individual desires for the good of the group as a whole. Hostility is the first resistance against the common good and serves as a defense against fear. There is fear of "exposure," fear of rejection by the leader, fear of competition with other group members for the affectionate attention of the leader, in addition to a multitude of unconscious fantasy-determined strivings. All the fears of the childhood family situation are mobilized. Basic to all these fears is the fear of and difficulty in managing one's tender feelings toward the leader and all group members. Here lie the shadowy forms of tender love and aggression. Certain group therapy techniques seem to be designed to avoid involvement in this kind of interaction. It seems true that human beings have more difficulty with their tender feelings than with their hostile ones. From this point of view the delicate position of the leader is crucial since it is through the leader that each member can move to more mature

relationships with other group members. The primary goal of any group is the attainment of group unity for the purpose of mutual exploration, analysis, and synthesis. The necessary pre-condition for mutual analysis and synthesis is the willingness to let oneself be close to others, motivated primarily by tender feelings to others.

INSIGHTS GAINED THROUGH GROUP THERAPY WITH MULTIPLE SCLEROSIS PATIENTS. ROBERT T. LONG, M.D.

Dynamic insights into the personalities of patients with multiple sclerosis were gained through the use of group therapy as a method of observation. Observations are based on a group of ten multiple sclerotic patients—5 men and 5 women. Psychologic and physical factors operated as selective factors. The patients were psychologically motivated. They were neither acute nor terminal cases, but in between these two extremes. The common factor of multiple sclerosis both helped and hindered the group process. This was of help in that the disease served as a common denominator and helped each member integrate more readily into the group. Their common disease hindered the group process in that it served as a strong resistance which was never resolved satisfactorily. Situations of anger or other painful emotions which arose in the group setting were either denied or produced withdrawal from the group. In explanation of this, attention is placed on functional concreteness of thinking which multiple sclerotic patients show. They must spend more mental energy thinking about physical acts which normal persons do automatically. It is further suggested that these patients show a disruption of the alarm mechanism of anxiety. Instead of serving a useful purpose, anxiety now only exposes them to greater danger, owing to the inco-ordination of their neuromuscular system. The necessity to avoid or deny stressful situations and emotions runs counter to the goal of therapy which is anxiety provoking. Because of this, interpersonal actions within the group setting were not resolved adequately.

THE GROUP BEHAVIOR OF MOTHERS OF SCHIZOPHRENICS. MAX DAY, M.D., AND IRVING ROSEN, M.D.

The observations in this paper were made by the authors in two ways: in a group of mothers of chronically ill schizophrenic women, and in a group of 4 chronically ill schizophrenic women together with their mothers. The following observations are applicable to "chronic" mothers and may not be true of mothers who give up the patients as hopeless early in the schizophrenic illness. In groups, these mothers presented the picture of individuals who felt helpless, unappreciated, and laden with guilt. As a result, they needed constant reassurance from the group members, their daughters, and the physician, of their worth. Comments on questions or disagreements confirmed for them their feeling that they were guilty and brought forth retaliatory denial of feelings of threats of separation. They seemed to want the leader's personal interest but not a close relationship,

one that dealt only with concrete physical details. Their guilt led to maneuvers to pin the guilt on someone and to constant activity to assauge their guilt. The activity might be their own or on the part of someone else, but it had to be on a concrete level to be acceptable to them. Closeness at all levels was important to the mothers, but it had to be concrete to avoid feeling involvement.

DYNAMIC INSIGHTS GAINED FROM GROUP THERAPY WITH OBESITY PATIENTS. STANLEY S. KANTER, M.D.

Comparison of "non-direction," and Id approach," and "Ego approach" on the part of the therapist revealed the development of a group climate marked by chaos, negative transference, and positive transference, respectively. Both sibling rivalry and the provision of emotional gratifications appeared to play roles in the weight loss. The obese group was marked by intensely ambivalent transference to the therapist and intermittent perception of the group as a primitive maternal super-ego. Anxiety was a prominent feature of the countertransference. Two-year follow-up yielded promising results.

Dynamically, the chronically overweight appeared to utilize obesity and overeating to shore up the defenses of an ego weakened by a noxious early family constellation. Intense ambivalence, isolation, and oversensitivity were particularly characteristic of these patients.

Discussion.—ELVIN V. SEMRAD, M.D.

These papers represent considerable and considerate effort in formulation of what goes on in group therapy. I compliment the authors on their sincere work and appreciate very much the opportunity to think with them about their observations and formulations.

Apropos Dr. Mann's paper, I agree that the setting of group therapy is one in which a group member is forced to be close to others, and I agree that the working through of hostility is a necessary preliminary to goal achievement by the group. I am not entirely sure that the specific fear is that of exposure. Could we consider this initial reaction as part and parcel of an attempt to adapt to this new situation, a situation of permissiveness, active interest in and by the leader, focusing on interaction, etc.? Is it possible that in this unique situation the members feel at a loss to attain satisfaction, at least immediately, usually protesting the nonstructured nature of the group with consequent negative feelings about which they are anxious? One of the characteristics of this anxiety is that members show constant expectation that the objects of an aggression will retaliate in kind. Some of our members have some (especially in teaching groups) ego skills in achieving closeness. These may not be considered by them appropriate to this new situation. To find a place in the group, or more correctly this group, requires a special skill which is not present, and, at least temporarily, interpersonal relationships become dissatisfying, so that the negative feelings have to be hidden, or at best expressed as hostility because the ego

capacity for useful self-assertion apropos of this kind of a situation is not fully developed. The awkward attempts at aggressive stands suggest this possibility.

The leader's function at this early period of a group's work is to help the group study hostile feelings and tolerate retaliation fears through identification and analysis in the group so that these can be dealt with in more mature ways. May we say he fills the more mature billets temporarily abandoned by the group members in meeting the overwhelming experience of this new situation? This situation is like any situation that a person has to meet with which he has had no previous experience.

This leads me to a discussion of Dr. Mann's statement that "all the fears of the childhood family situation are mobilized." There is no question in my mind that this may be the essence of the dynamics, but it prompts me to think further in terms of manifestations which may lead one to suspect that later group experiences may be recapitulated here. Sometimes we observe concerns of prepubertal nature especially in the more mature groups, especially where group unity becomes more a matter of finding "appropriate distance" for effective negotiation with each other. I do not know whether this is what Dr. Mann implied when he said that the "precondition for mutual analysis and synthesis is a willingness to let oneself be close to others, motivated now primarily by tender feelings to others." "Appropriate distance" closeness minimizes anxiety to the point where libidinal forces are minimally threatening and can become useful in the process of creating; "learning in the academic sense" and "making changes for the better" in the therapeutic sense.

With regard to Dr. Long's paper, I was interested in his statement, "This denial of even the slightest hostility has been shown by each member of the group and has not as yet been satisfactorily resolved." Likewise I was interested in his studies of the multiple sclerosis patients' sensory and motor deficits. I would like to ask him if he really believes that this is what makes denial so necessary, or whether he has in his experience seen patients use their inconveniences occasioned by these deficits to bid for affection from other people. A person who feels deprived (here also deprived of his equipment) finds great difficulty in attaining satisfaction in interpersonal relationships because not only in fact, at least in fancy, he is deprived and rejected and consequently has a paralyzing uncertainty as to the affection and respect in which he is held. He thus may burst out in awkward and ungracious self-assertions which are difficult for his objects to consummate. To deny and hide becomes most necessary. Besides he cannot take a chance on losing more than he has lost already, because he does not know who or what caused his illness or who or what will make it worse, that is, when will the object of an aggression retaliate in kind? I have a hunch when we will learn more about how to supply affection to those who must deny that they need it that the importance of the motor sensory deficits in terms of psychologic adaptation will become less, and I wonder if Dr. Long thinks so too.

Dr. Day's and Dr. Rosen's paper gives me a chance to say a word about what I consider the most therapeutic ingredient for dealing with denial. We casually talk about supplying affection, being permissive, etc., but we too infrequently stress the importance of whether our patients feel we really care in the full sense of the word for them by helping them identify, tolerate, and eventually meet and adjust to the realities of their emotional lives and relationships. The leaders captured the interest of the mothers when they became "active, provocative, and direct," apparently genuinely letting the mothers know that they had some very definite feelings about them. Their statement, "Yet with all, the doctors really like them," makes me ask if this affective ingredient couldn't have accomplished the same as the former since it was sincere, genuine, and betrayed the feeling that the patient "really mattered" to the group leader. I would like to ask a question apropos the statement, "This useless energy used in frantically trying to do the doctor's job was later in the year channeled for some of the mothers into talk and plans for working toward better mental health conditions on a state level." My question is, were these energies channeled to perform a mission because they could not be analyzed and utilized in helping the mothers be less difficult for their daughters to live with?

Apropos Dr. Kanter's paper, I surmise that he has found the ego approach the most effective. I cannot attest to the dynamic insights gained from group therapy of obese patients, as I have never had the experience of participating in such a group. However, from other sources of information, I would agree that the observations he made are relevant.

BOOK NOTES AND REVIEWS

Books received are acknowledged and given a brief description in this section. Selections serving a variety of interests will be made from these for a more extensive review, as far as space will allow.

BOOK NOTES

Borberg, Allan. CLINICAL AND GENETIC INVESTIGATIONS INTO TUBEROUS SCLEROSIS AND RECKLINGHAUSEN'S NEUROFIBROMATOSIS. [Copenhagen: Ejnar Munksgaard, 1951. Pp. 239.]

This excellent study was made during the years 1944-49 in collaboration with the University Institute for Human Genetics in Copenhagen. The material which is rare was collected from several institutions.

The historical contributions, the theoretical aspects, the incidence, symptoms, forms, course, pathology and the genetic findings of these two syndromes have been well presented in a succinct and orderly fashion. There are numerous points of resemblance between the two disorders. They both suggest a developmental anomaly affecting the neuro-ectoderm. Also in both conditions there are glial processes, fibromatous skin changes and pigmentary abnormalities. Both conditions occur as hereditary phenomena, but there are still differences of opinion among investigators as to their relationship.

The histories and pedigrees of 37 tuberous sclerosis families are presented under the captions of "unquestionable inheritance", "questionable inheritance" and with "no signs of inheritance." The same arrangement has been followed for 83 Recklinghausen families. These case reports contain a great wealth of information and there are 483 bibliographical references to publications that have accumulated in the literature over many years.

This unique thorough-going investigation may be highly recommended to neurologists and pathologists.

Gehry, K. KORPERKRANKENPFLEGE, Band I (Zweite Anflage). [Bern: Hans Huber Verlag, 1950. Pp. 272 with 16 colored plates and 130 figures. Pr. Fr. 27.50.]

This is the second edition of a book that should be very useful to the nurse and others who care for physically ill patients. It begins with an account of atoms, cells, tissues, and inheritance in their roles in body structure, continues with descriptions of general disease processes such as inflammation, swelling, fever, etc. and passes on to the anatomy, physiology, pathology, and clinical disorders of all of the organs and organ systems.

There is a special section on infectious diseases and wound infections, one on the particular features to observe in an ill person such as pulse, weight, skin, urine, etc. and an extensive section on practical methods and procedures in diagnosis, therapy and general care.

There is an excellent brief chapter on first aid methods for several types of emergency and a final short account of dying and death.

The text is liberally illustrated throughout by clearly defined photographs and drawings. It is a book well worth having in the hospital library and as a text book for nurses.

Stern, Erich. Lebenskonflikte Als Krankheitsursachen: Eine Einfuhrung in die Psychosomatische Medizin. [Zurich: Rascher Verlag, 1952. Pp. 360. Pr. Fr. 21.]

In this book we have another coverage of the field of psychosomatic medicine which is becoming ever more popular. The author, after introductory sections on concepts and methodology, describes as is customary in such texts, disorders of the heart, respiratory organs, female and male genital disturbances, disorders of the gastrointestinal tract, of urination, of the skin, of motility, of the sense organs and of the endocrine and metabolic systems.

The special topics of syphiliphobia, aging, senility and death, psychosomatic medicine in surgery, and in pediatrics are of particular interest. A discussion of psychic factors in tuberculosis occupies a separate section. This disease is apparently creating a good deal of interest psychosomatically in other countries as well as in America. Heinrich Heubschmann devoted a whole book on this subject (1952) published in Stuttgart.

Dr. Stern presents the ideas and contributions of many authors in psychosomatic areas constituting a useful review of the literature. His book is psychodynamically oriented and thus modern in approach and outlook. It is well packed with facts and concepts.

The text is completed by a general summary of psychic factors in disease and by a presentation of some therapeutic inferences and conclusions. At the end there is a liberal list of references, and an author and subject index.

Huebschmann, Heinrich. Psyche und Tuberculose. [Stuttgart: Ferdinand Enke Verlag, 1952. Pp. 284. DM 22.80.]

For many years any relationship, that may exist between tuberculosis and the mind has been the focus of much discussion and comment, and of some worthful studies.

Dr. Huebschmann of the Heidelberg Clinic has approached the problem from a wide comprehensive view point, the details of which he has presented in terms of the fundamentals of the general and special pathology of the disorder as it affects different aspects of the physical

organism, and in the second part of the book in terms of mind and disease.

The various constitutional factors, the relationship of mind and pathogenesis in tuberculosis, the organ pathology, and the way these several systems are integrated in the overall picture of the disease are discussed in an interesting account which represents psychosomatic medicine as it should be investigated.

The text has been skillfully organized and anyone using the table of contents can quickly locate what he seeks. The third part of the book is a succinct summary of the problem and the findings. German reading internists and psychiatrists will find it interesting material.

Leonhard, Karl. GRUNDLAGEN DER NEUROLOGIE. [Stuttgart: Ferdinand Enke Verlag, 1951. Pp. 313 with 40 illustrations. Pr. DM 18.80.]

In 1948 Prof. Karl Leonhard of Universitat Frankfurt am Main, published his *Grundlagen der Psychiatrie* which has served as a handy text for the German speaking students. The present book in accordance with its title deals with neurological problems in terms of their basic principles.

The three divisions of the text are Part I presenting the classical neurological examination, Part II which describes the symptomatology of the different neurological systems and Part III which presents the neurological disease entities usually covered in the text books; that is, those characteristic of peripheral nerves, spinal cord, cerebellum, cerebral hemispheres, scleroses, infections, intoxications, traumatisms, vascular disorders, tumors and in addition, sections on avitaminosis, insolation, electric current injuries, and vegetative nervous system disorders.

A lot of ground has been covered for a book of this relatively small size, therefore the descriptions are brief. It is a useful book for the orientation of students in this field.

Sjögren, T., Sjögren, H., and Lindgren, A. G. H. MORBUS ALZHEIMER AND MORBUS PICK: A GENETIC, CLINICAL AND PATHO-ANATOMICAL STUDY. [Copenhagen: Ejnar Munksgaard, 1952. Pp. 152 with 11 illustrations.]

This monograph deals with a large representative material on these comparatively rare diseases. The genetic, clinical and pathological studies were carried out in accordance with a special grouping of the cases: Group one, consisting of 18 probands (12 women—6 men) were the histopathologically examined cases of Alzheimer's disease; Group two, 18 probands (11 women—7 men) was composed of Pick's disease cases, histopathologically examined; Group three, 29 probands (22 women—7 men) contained cases not histopathologically examined, but diagnosed clinically as Pick-Alzheimer Syndrome, the cerebral atrophy, verified either by encephalography or autopsy, and group four composed of 15 probands (13 women—2 men) diagnosed clinically as

Pick-Alzheimer Syndrome without verification by histopathology, encephalography or autopsy.

The genetic, clinical and pathological analyses with reference to this grouping have been exceptionally well done, and the differentiation of these disorders from senile dementia seems quite convincing. The important differences are discussed. The monograph contains tables, pedigree charts and photographs of the characteristic cerebral pathology. It is a very valuable addition to the literature and knowledge of the disorders covered.

Thompson, Geo. N. The Psychopathic Delinquent and Criminal. [Springfield, Ill.: Charles C. Thomas, 1953. Pp. 157, illustrated. $4.50.]

Psychopathic delinquents and criminals comprise the group of law breakers that have been most difficult to diagnose correctly, treat successfully and integrate into the life of human society. Most writers on this subject are far from clear in the meaning of their terms. "Constitutional psychopath", "psychopathic personality", 'psychopathic delinquent" and "psychopathic personality with psychosis", etc. are names conveying different concepts to different people, in fact, some workers advocate throwing all such terms out of use. The author of this book has fortunately defined the terms "psychopathic delinquent" and "psychopathic personality" as they are used in the text and has made a good job of it. After all they are really valuable terms referring to rather definite behavior patterns and should be retained and used correctly.

Following definitions and clarification of terms, subsequent chapters consider in order, etiology, evidence of organic factors, question of environmental causative factors, the psychopathic personality, psychopathology, course of the disorder, delinquency due to other psychiatric abnormalities and finally treatment.

Psychiatrists, neurologists, psychologists, social workers, sociologists and those associated with courts of law should read this little book carefully since it deals wholesomely and constructively with a type of individual poorly understood in general and badly handled in particular by society.

BOOK REVIEWS

Schaltenbrand, Georges. Die Nervenkrankheiten.. [Stuttgart: Georg Thieme Verlag, 1951. Pp. 880, 539 illustrtions. $20.70.]

The author who undertook to write a new textbook on neurology, is in the fortunate position to have trained in several countries, particularly Germany and the United States. This, undoubtedly, widened his view, and it should be acknowledged that the book gives full credit to the American contributions to neurology. It is a truly international volume on neurology and the only shortcoming is that

the references since 1940 are somewhat spotty, obviously due to unavailability of the non-German literature during the immediate postwar period in which the book was finished.—It is difficult to review a book which actually represents the second part of a textbook whose first part on general neurology is not yet available. The nosological part dealt with in this volume is a contribution to the neurological literature which can only be highly recommended. It is inevitable that the reviewer compares any new German textbook on neurology with the old classic by Oppenheim. This will be possible only when the whole opus is available but the present volume stands the comparison well, although it is more modest in its purpose. Oppenheim's book had the size of a textbook but came very close to a handbook with its many references. Schaltenbrand lists references in the text only as far as the most important contributions on the subject under discussion are concerned. This limits the scope of the book but it makes reading easier and contributes to its usefulness for the practicing neurologist. He will find all the important information as far as the disease entitles are concerned, and occasional omissions seem to concern points which will be probably found in the still missing parts on general neurology. Since the more recent contributions to neurology were mostly made in the field of neurophysiology, it is of advantage and probably on purpose that the general part will appear at a later date when more references from the last decade will be available to the author.

Of points to be mentioned in a critical review, a definite neurosurgical orientation and knowledge is one of the main advantages. The feeling of many that neurology is losing ground to neurosurgery is best counteracted by the effort on the part of neurologists to remain abreast of neurosurgical viewpoints and developments. Relationships to internal medicine, orthopedics, dematology and other medical fields find an equally thorough discussion. Surveying such a comprehensive study is a welcome opportunity to be reminded of the great variety of neurological conditions and their relationship to and importance for most other specialties.

An important place is given to a thoughtful consideration of therapeutic possibilities. The lack of a real cure in so many neurological conditions is undeniable but a therapeutic nihilism as an attitude is successfully avoided by the author. A full appraisal of Schaltenbrand's textbook must wait for the parts on neurological examination, pathophysiology and syndromes. The present volume whose carefully chosen and well reproduced illustrations should be mentioned specifically, can only be highly recommended. Its translation into English would be desirable. LOTHAR B. KALINOWSKY.

Von Foerster, Heinz, Mead, Margaret, and Teuber, Hans Lukas. (eds.) CYBERNETICS: CIRCULAR CAUSAL AND FEED BACK MECHANISMS IN BIOLOGICAL AND SOCIAL SYSTEMS. Transactions of the Eighth Con-

ference, March 15th, 16th, 1951. [New York: Josiah Macy, Jr. Foundation, 1952. Pp. xx + 240. $4.00.]

This well-printed volume is the third in a series dealing with cybernetics; and records the verbatim and mainly extemporaneous exchange of free discussion and debate (from stenographic or recordings) engaged in by some twenty-two noted representatives of various disciplines (biological, mathematical, engineering, psychological, etc.) on the topic of communication. The goal of these symposia is "the promotion of meaningful communication between scientific disciplines". The discussions are free and informal. They are, in marked contrast to the usual (and often useless) scientific gatherings, not designed to present pre-conceived, rigid and closed solutions to "tidy problems", but to elicit really provative discussion of the difficulties met in research, practice, and organization in every discipline. As the editors make clear "these pages should rather be received as the partial account of conversations with a group". It thus "attempts to recapture a fragment of group interchange in all its evanescence and represents one of the few concerted efforts at interdisciplinary communication". The present discussion revolves around communication and self-regulating mechanisms.

For a period of six years the association of some twenty-five experts in various areas has been maintained in a series of seven symposia. The present volume covers topics such as communication patterns in problem-solving groups, communications between men (meaning of language), between sane and insane (hypnosis), between animals, presentation of a maze-solving machine (Shannon's mechanical rat), search for basic symbols, and the nomenclature of information theory. From the very nature of the meetings no real idea can be conveyed in a brief review of what was accomplished. No hard and fast conclusions reached. But for fascinating, stimulating, and provocative reading this book would be hard to beat. There is a clash and flash of highly intelligent and acutely trained minds maintaining a very high level of understanding. A particular note must be made of the groups concern in the exploration of the communication actually taking place among themselves. The enormous difficulties of inter-disciplinary action, understanding and communications is nowhere else so vividly portrayed.

No one interested in the most advanced ideas in many disciplines, as well as the progress in multi-disciplinary collaboration, should neglect these and other series of publications being issued by the Macy Foundation. This is a real pioneer effort and deserves our support. There is only one sour note: the use of highly glazed, acetated paper of this volume is a torture for the eyes of most readers. It should be avoided if at all possible. The ophthalmologists have long inveighed against it.

JOSEPH ZINKIN.